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ANALYSIS OF SHOCK

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During a period of 40 years shock has been the subject of extensive and intensive study, both clinical and experimental. This has resulted in a mass of well-attested factual evidence, but agreement on the pathophysiological mechanisms has not been reached. Writers differ widely in their views, and readers are bewildered by the evidence set forth in support of the conflicting opinions. It appears that a logical correlation of the known facts is needed now rather than a search for new facts. A realistic examination of the causes for disagreement may be illuminating.

What is Shock?

The broad and indefinite use of this term is not the least among several causes for disagreement. *Shock* has been applied to a number of unrelated conditions. Many regard it as a state of acute circulatory failure characterized clinically by prostration, apathy or stupor, tachycardia, feeble regular pulse, and diminished blood pressure. The peripheral temperature frequently is subnormal. When used in this sense shock includes several forms of circulatory disturbance having no other features in common; their pathogenesis and disorders of function are various. The effects of syncope, fright, exhaustion, anaesthesia, haemorrhage, primary shock, cardiac failure, or of secondary shock may produce the clinical signs mentioned. Each of these conditions may cause low blood pressure; hence it cannot be used as the criterion for differentiating them.

The attention of surgeons is focused chiefly upon shock seen in battle casualties and in those injured in accidents or air raids, and upon shock following extensive surgery. If, under these or other circumstances, a patient manifests the signs mentioned above, shock is regarded as present. Some writers recognize that at least *three unrelated mechanisms* may combine to cause weakness, pallor, feeble rapid pulse, and low blood pressure —interpreted clinically as shock—after traumatic injuries. Attention is invited to a brief consideration of each of these mechanisms.

Primary or Neurogenic Shock.—This is a neurovascular reaction like that of syncope or fainting. It may be excited by pain, fear, and the emotional effects of the injury, or by non-sensory nerve impulses arising in the damaged tissues. Primary shock comes on promptly, and is usually transient unless accompanied by extensive trauma or haemorrhage. In cases of severe abdominal injury, visceral perforations, pancreatitis, burns, or extensive damage to other parts, primary shock may merge gradually into secondary shock without an interval of partial recovery. This feature was emphasized by Cope (1928, 1935), Blalock (1940), and others.

Recent experiments (Phemister and associates, 1944) show that prolonged low arterial pressure can be produced in animals by continuous mild stimulation of afferent depressor nerves. Arterial pressures between 40 and 60 mm. Hg were maintained in some instances for several hours. The blood of such animals showed dilution regularly. This feature is opposite to that seen in secondary shock. It indicates that the mechanism of fluid balance is functioning normally and that the capillary walls are not abnormally permeable. Fluid balance and haemoconcentration will be discussed later. A decrease in total blood volume has not been shown in uncomplicated primary shock.

Haemorrhage.—The effects of haemorrhage are an obvious cause for low blood pressure and other signs of disturbed circulation after injuries. These signs may develop promptly from voluminous haemorrhage or gradually after slow or repeated small haemorrhages. The clinical signs of haemorrhage are like those of secondary shock, but they differ in other important particulars, as will appear presently. Haemorrhage directly lowers the total blood volume and is followed by a rapid dilution of the blood. This is accomplished by absorption of fluid from the tissues into the blood, tending to restore the blood volume. It is evident that the mechanism of fluid balance is operating physiologically. Low blood pressure developing shortly after trauma is due chiefly to neurogenic and haemorrhagic effects.

Secondary Shock.—This is the third mechanism by which circulatory deficiency may develop after extensive injuries. It is due to atony and abnormal permeability of capillary walls, and has been discussed (Moon, 1938a, 1942a, 1942b) as circulatory failure of capillary origin. Products of tissue autolysis or of infection, absorbed from damaged tissues, produce permeability of endothelium like the effects of histamine, peptone, venoms, and other capillary poisons. When this effect is produced in extensive areas it involves the mechanism of fluid balance. Osmosis requires the presence of a semi-permeable membrane—the endothelium. If the latter becomes so permeable that plasma colloids escape through it, osmotic attraction ceases to act and the mechanism of fluid balance is vitally deranged.

Leakage of fluid from the blood into the tissues lowers the blood volume and causes haemoconcentration, which is evidence in itself that the mechanism of fluid balance is not functioning adequately. Under normal conditions fluid lost from the blood is restored by absorption; haemoconcentration is thereby prevented.

Decreased blood volume, combined with increased volume capacity of the capillary bed, tends to cause circulatory deficiency, which is manifested by clinical signs like those of primary shock or the effects of haemorrhage. But the mechanism of capillary damage requires time for development; it is never seen immediately after injury, hence it is called *delayed* or *secondary* shock.

Confusion in Shock Analysis

Numerous reports, both from British and from American authors, indicate confusion in attempting to analyse the causes of shock resulting from injuries. Space does not permit detailed examination of each of these. Since the reports are similar in nature and in the conclusion drawn, one will be cited as an example.

Evans and his associates (1944) made haematocrit and blood-volume determinations in 143 cases of knife wounds, bullet wounds, and accidental injuries. Many of these developed shock, as indicated by low blood pressure and other clinical signs. Haemodilution was present in the majority; a few showed haemoconcentration. The authors concluded that low arterial pressure is the most useful criterion for shock and that haemorrhage is its most important cause. It was stated that the injured were seen *soon* after the wounds were received. Hence probably the time factor was not sufficient for the development of secondary shock. In many instances there was haemodilution, and evidence of extensive haemorrhage which averaged 38% of the estimated blood volume. It is probable that primary shock and haemorrhage were chiefly responsible for the low blood pressure.

However, among 63 cases of moderate or severe shock from skeletal, thoracic, and abdominal injuries there were 46 instances in which the haematocrit reading was 40 or above. Do the authors believe that haemorrhage was the major factor in *these cases*? A haematocrit reading of 40 indicates approximately 90% haemoglobin or 4,500,000 erythrocytes. Those who have lost 38% of their blood volume by haemorrhage should show haemodilution of greater degree than this. One suspects that some other mechanism was operating in this group of cases (see below: Haemoconcentration). Evident confusion is expressed in the authors' frank statement: "In his search for truth concerning traumatic toxaemia the reader may become bewildered by some of the writings on the subject."

The reasons for bewilderment are not far to seek. Conditions due to a combination of causes are not suitable for analysis of the mechanisms involved. By way of illustration:

Let X symbolize the effects of primary or neurogenic shock
" Y " " " haemorrhages, " haemorrhagic " shock
" Z " " " capillary damage, " toxaemic " shock
Then X + Y + Z = traumatic shock (as manifested clinically)

To propose conclusions as to the cause of shock upon data from cases of traumatic injuries is as unsafe as to propose the solution of a single equation containing three indeterminate variables.

Circulatory deficiency following trauma may be due to neurovascular reactions or to the effects of haemorrhages or to the absorption of toxic products, or it may be due in part to each. So-called *traumatic shock* is not a disease entity but a syndrome; it represents the summative effects of several factors. These vary in different cases and in the same case at different times. Obviously, the mechanisms involved in shock from trauma should be studied separately as neurovascular, haemorrhagic, and toxaemic factors.

Secondary Shock and Haemorrhage

Experimental studies on shock from trauma have been confused by the same combination of indeterminate variables as those present after injuries. Such experiments include also another item, the anaesthetic, which tends to lower the arterial pressure. A method commonly used experimentally is to narcotize an animal deeply with barbital or some other anaesthetic agent. Shock is then induced by extensive trauma to the muscles, by intestinal manipulation, or by some other form of tissue abuse. Variations in the arterial blood pressure serve as the chief criterion of the animal's condition. Attention has been directed repeatedly (Moon and Kennedy, 1932; Moon, 1938a, 1942a, 1942b) to sources of error inherent in this mode of experimentation. Workers did not provide controls on the factors of anaesthesia and of haemorrhage. Narcosis with barbiturates and with other agents often causes low blood pressure.

Variable amounts of blood are lost owing to the trauma. In some reports the workers showed convincing evidence that they were dealing almost entirely with haemorrhage. Several hours' time is required for autolysis and absorption from damaged tissues to produce systemic effects. The animals often succumb to the effects of haemorrhage and anaesthesia before this occurs. Under such conditions the observed results may be due to narcosis or to haemorrhage or the absorption of toxic products, or they may be due in part to each. Manifestly it is unsafe to base conclusions as to the relative effects of haemorrhage and of secondary shock upon data obtained from experiments of this kind.

The necessity for studying each item separately is re-emphasized. Primary shock has been investigated clinically and experimentally by Phemister and his associates. A study of the effects of simple haemorrhage without anaesthesia or trauma presents no difficulties. We have reported such an investigation (Moon *et al.*, 1941). Circulatory failure due to capillary atony may be produced by injections of histamine, extracts of normal tissues, bile, cholic salts, peptone, bacterial toxins, venoms, or other capillary poisons. Several of these agents are concerned in the development of shock incident to intoxications and infections in man; but, excepting tissue extracts and bacterial toxins, they are not substances which usually result from autolysis and bacterial growth in traumatized tissues. The autolysis of tissues *in vivo* (Moon, 1942a) provides an experimental method for observing the effects of absorption, uncomplicated by anaesthesia and haemorrhage.

One method is to introduce a quantity of finely minced fresh normal tissue into the peritoneal cavity of a normal dog. Light ether anaesthesia is used while the abdomen is opened and the tissue, suspended in saline solution, is inserted. Recovery from the anaesthesia is prompt; the animal behaves normally for an hour or two, then progressive illness accompanied by vomiting develops. The blood becomes concentrated, the pulse rate increases, the peripheral parts become cold, urination is suppressed, and death by circulatory failure occurs 8 to 36 hours later. The severity of the illness and the speed of its progress depend upon the amount of tissue used. A dosage of 5 g. of liver or muscle pulp per kilogramme of body weight often results fatally in 8 to 10 hours; marked illness but eventual recovery may result from a dosage of 1 to 2 g./kg. The rapidity and severity of the illness are increased under anaesthesia as used for recording blood pressure.

The results described are highly significant for two reasons: they indicate that products derived from the autolysis of normal tissues will cause death by circulatory failure like that of secondary shock; also, the associated physiological disturbances and the changes seen in the viscera were identical with those resulting from the injection of capillary poisons and from anaphylactic shock.

A distinctive pattern of circulatory changes is seen when animals have died of shock caused either by autolysis *in vivo* or by the other agents mentioned above. This pattern consists of diffuse capillo-venous hyperaemia, oedema of soft tissues, capillary haemorrhages in mucous and serous surfaces, sometimes effusions of fluid in serous cavities, and parenchymal degeneration and necrosis. Such changes occur in the thoracic, abdominal, and cranial viscera; they are not seen in the external or somatic tissues.

The same pattern of visceral changes is seen when secondary shock has been the major cause for death in man. These changes are in sharp contrast to those resulting from haemorrhage. It appears that no one has compared carefully the physiological disturbances resulting from haemorrhage and those from secondary shock. My associates and I undertook such a comparison. We collected the recorded observations of different workers on various features seen in these conditions; then we verified each item experimentally (Moon *et al.*, 1941). The results of these comparisons follow:

Features Identical in Shock and Haemorrhage

Sympatho-adrenal activity
  Stimulation of myocardium
    Strong rapid pulse in early stages
  Peripheral vasoconstriction
    Reduced volume flow
    Peripheral ischaemia; pallor
    Loss of tissue turgor
  Discharge of reservoir blood into systemic circulation
    Contraction of spleen
  Increased blood sugar
  Dilatation of pupils; often perspiration
Low basal metabolism
  Declining temperature
Decreased alkaline reserve
Increased respiratory rate; thirst
Low arterial blood pressure (in late stages)
Death due to inadequate circulatory function

Because of these similarities shock and the effects of haemorrhages have been regarded as identical. Other features which have been reported in secondary shock from wounds, burns, intestinal obstruction, anaphylaxis, and transfusion reactions are tabulated below and compared with the same items after simple haemorrhages.

We have attempted to produce the features recorded under the heading of Shock by haemorrhages of all degrees and varying duration, by successions of large haemorrhages ending fatally in 4 to 8 hours, and by repeated small haemorrhages with death in 1 to 4 days. In every instance the findings recorded under Haemorrhages resulted; in no case did the changes characteristic of secondary shock develop.

It seems remarkable that such numerous and important differences escaped the attention of those who have written voluminously on haemorrhages as the cause for shock. More remarkably, a few even advocate haemorrhages as the method of choice for producing shock experimentally. Let it be emphasized that haemorrhages, when present, are a highly important contributory factor; an animal or person in shock may not survive a minor

haemorrhage which under normal conditions would have insignificant effects. But haemorrhages alone, independent of trauma, anaesthesia, or toxic effects, will not produce the syndrome of secondary shock.

Contrasted Features

Items	Shock	Haemorrhages
Endothelium	Permeable to colloids	Impermeable
Flow of lymph	Increased	Decreased
Tissue fluid	,,	,,
Fluid balance	Disturbed	Undisturbed
Absorption	Impaired	Unimpaired
Vomiting	Persistent	No vomiting
Diarrhoea	Frequent	Absent
Saline solutions, intravenous	Ineffective	Often effective
Renal:		
Excretion	Deficient	Unimpaired
Urine	Concentrated, low volume, albumin, erythrocytes, bile, debris	No characteristic changes
Blood:		
Coagulation time	Lengthened	Shortened
Concentration	Increased	Decreased
Non-protein nitrogen	,,	,,
Potassium	,,	Terminal increase
Plasma chlorides	Decreased	Increased
Necropsy findings:		
Oedema of soft tissues	Characteristic	None
Serous effusions	Present	Absent
Capillo-venous congestion	Characteristic	,,
Petechiae	,,	,,
Visceral ischaemia	Absent	Present
Organ weight	Increased	Decreased
Gastro-intestinal tract	Dilated, atonic	Contracted
Parenchymal necroses	Present	Absent

Circulatory Dynamics

The principles of capillary physiology (Starling, 1909; Ebbecke, 1923; Lewis, 1927; Krogh, 1929) serve to explain how various noxious agents produce circulatory deficiency. A diversified group of substances which affect endothelium deleteriously are known as capillary poisons. These include extracts of normal tissues, products of tissue autolysis, foreign proteins and protein-split products, bacterial substances, histamine, bile, venoms, poisons, metabolic products, and even moderate lack of oxygen. It was established that any kind of injury to endothelium increases its permeability to the plasma colloids. Thereby other dysfunctions result as shown in the following table:

Agents or conditions injurious to endothelium	Pathological effects:
	Endothelial permeability
	Disturbance of fluid balance
	Oedema of soft tissues
	Serous effusions
	Increased flow of lymph
	Haemoconcentration
	Capillo-venous congestion
	Stasis
	Petechial haemorrhages

Permeability of capillary endothelium lowers both the actual and the effective blood volume. Leakage of plasma into the tissue spaces reduces the *actual* blood volume and causes haemoconcentration, while sequestration of blood by stasis in dilated capillaries and venules reduces the *effective* blood volume. This combination leads to a disparity between the volume of blood and the volume capacity of the vascular bed. Such a disparity produces a circulatory deficiency superficially resembling that resulting from haemorrhages. Physiological reactions compensate for minor degrees of such a deficiency. Activity of the sympathoadrenal system causes arterial constriction, stimulates cardiac function, and causes the spleen to contract, discharging its reserve of blood into the circulation. Selective distribution of blood favours the vital organs at the expense of non-vital parts. Peripheral circulation is reduced, and the external parts become pale, cold, and almost pulseless.

So long as this compensation is effective there is no marked decline in the arterial blood pressure, but the latter is maintained at the expense of the volume flow. Stagnation of circulation in the dilated capillaries reduces the amount of venous blood returned to the heart. This lessens the cardiac output and decreases further the volume flow. A reduced delivery of oxygen to the tissues follows, with accompanying anoxia, which of itself causes further relaxation and permeability of capillary endothelium (Krogh, 1929; Landis, 1934, 1937). This feature introduces a self-perpetuating factor into the mechanism, causing it to progress in a vicious circle. When finally the compensatory reactions are no longer adequate the blood pressure declines progressively and the complete syndrome of shock is manifested clinically. The items which constitute that syndrome were tabulated above in the comparison between shock and the effects of haemorrhages.

Circulatory deficiency originating in the capillaries produces the pattern of visible changes in the viscera described in a preceding section. The capillaries and venules become relaxed and distended with closely packed corpuscles, stagnation of circulation develops; stasis is the surest sign of increased capillary permeability (Landis, 1934, 1937). The leakage of plasma causes oedema of soft tissues and effusions into serous cavities. Dissolution of capillary walls causes minute haemorrhages in mucous and serous surfaces and in parenchymatous tissues. These changes are seen in varying degrees when secondary shock from any cause has been the major factor leading to death.

We have noted that marked hyperaemia and petechial haemorrhages predominate when severe shock has led to death in a few hours. Serous effusions and oedema are prominent when the deficiency has developed more gradually, leading to death after 24 hours or longer. We were able to produce pulmonary oedema experimentally (Moon and Morgan, 1936) by burns of moderate extent, by tissue autolysis *in vivo*, and by injections of various capillary poisons. To accomplish this it was only necessary to decrease the dosage of the agent so that fatal circulatory failure might develop more slowly. Partial circulatory stasis and oedema of the lungs predispose to pneumonia. We have reported (Moon, 1938b) on secondary pneumonia as a terminal event in shock of sublethal degree.

Half-truths

Erroneous beliefs persist longer when supported in part by truth than when entirely false. Propaganda is far more effective when based upon a modicum of fact; likewise in scientific matters. One example has been seen in the belief that shock from trauma is due entirely to haemorrhage. Another instance is the interpretation that leakage of plasma locally is *the* cause for low plasma volume.

The decreased plasma volume which develops after burns is attributed by many writers entirely to capillary leakage in and about the burned area. Scant attention is paid to the fact that capillo-venous hyperaemia and oedema develop in extensive visceral areas *remote from the burn*. Long ago it was shown (Schjerning, 1884; Bardeen, 1896-7, 1897; Pack, 1926) that the brain and meninges, the lungs, gastro-intestinal tract, liver, and kidneys are markedly hyperaemic, that the soft tissues are oedematous, and that petechial haemorrhages are found in the serous surfaces, mucous membranes, and parenchymatous tissues of the viscera.

These changes are the characteristic pathological finding after death from burns. Yet some ignore them, unwilling to admit the systemic effects of substances absorbed from burned tissues. It is true that local loss of fluid occurs about injured tissues; but it is equally true that such loss occurs also in visceral areas after extensive burns or trauma. Local fluid loss is only part of the mechanism by which plasma volume is reduced.

Some writers attribute the visceral hyperaemia and oedema to anoxia, and explain that they occur during the agonal period just before death. Clinicians who have listened to the breathing of severely burned patients testify to the presence of congestion and oedema when shock is present *before the agonal stage is reached*. It is true that anoxia will cause capillary atony accompanied by hyperaemia, stasis, and oedema. But this is not a complete picture of the relationship. Capillary atony and anoxia have reciprocal effects: either of them acting primarily will bring about the development of the other. This relationship gives the disturbance a self-perpetuating quality which operates in a vicious circle.

So long as the physiological mechanism of fluid balance is functioning, local fluid loss is compensated by the absorption of fluid from the tissues elsewhere. This maintains the blood volume and concentration within normal limits. For example, large amounts of fluid may be lost from the blood locally, as in hydrothorax or ascites, yet neither shock nor haemoconcentration follows. It is my belief that haemoconcentration is evidence of capillary permeability sufficient in extent and degree to disturb the mechanism of fluid balance. This may at times result from the *local* effects of burns or trauma of great extent. It probably will not develop from limited injuries.

Any loss of blood or fluid is of importance when secondary shock threatens to develop. The importance of the local loss is in proportion to the amount of fluid loss. The explanation of low plasma volume and haemoconcentration as resulting from local fluid loss is another instance of a part-truth.

Traumatic Toxaemia

During World War I the British Medical Research Committee organized a co-ordinated clinical and experimental investigation of shock following wounds. Eminent British, French, and American workers studied the manifestations seen in injured men and in experimental animals. These reports led to the theory known as *traumatic toxaemia*. It is summarized as follows:

> "The theory of secondary shock which has the strongest support, both in clinical observations and in laboratory experiments, is that of a toxic factor, arising from damaged and dying tissue and operating to cause an increased permeability of the capillary walls and a consequent reduction of blood volume by escape of plasma into the lymph spaces. Thus the concentration of the corpuscles is also readily explained. It is recognized that, after a sufficient time, infection may occur and be of such character in itself as to induce a persistent low blood pressure. According to this theory there may be no essential difference between the effects of toxins given off by damaged tissue and of toxins resulting from activity of bacteria." (Med. Dept. of U.S. Army in the World War, 1927.)

The chief objection to this explanation is negative in character, based on the fact that no toxic substances had been shown experimentally or identified chemically in the blood during shock. Digressing for a moment, let it be noted that the same objections and logic apply equally to the bites of venomous snakes. No toxic substance has been identified chemically in the bitten animal's blood; I have seen no report on transfusion of blood from a bitten animal to a normal one; it is highly improbable that the amount of venom so transferred would cause notable effects. The bitten limb swells enormously and its tissues are distended with blood and fluid. May not the effects of a snake-bite be due entirely to local loss of blood and fluid in the area affected? This explanation would make unnecessary the assumption that a toxic substance produces systemic effects after snake-bites.

Presumably the toxic substances which result from trauma are cleavage products of tissue autolysis or of bacterial action upon tissue proteins. Chemical methods are not yet perfected to identify such substances in the blood, except as non-protein nitrogen. The latter is markedly increased during shock, and is attributed (Whipple and van Slyke, 1918; Whipple *et al.*, 1920) to destruction of the body's proteins. The objection mentioned took no account of these facts nor of the mass of clinical and pharmacological evidence indicating toxic effects.

Recent reports indicate toxaemic effects in experimental traumatic shock. Best and Solandt (1940) instituted exchange transfusion of blood between pairs of dogs, in one of which shock was induced by trauma. The untraumatized dogs developed shock, as did those traumatized. These results were interpreted as indicating the effects of a toxic substance released from injured tissues and conveyed by the blood. Kendrick, Essex, and Helmholz (1940) performed a somewhat similar experiment by exchange transfusion, which likewise furnished evidence supporting the theory of toxaemia. Even those who have opposed vigorously the conception of a toxic factor have now relaxed their opposition. For example, Freeman and his collaborators (1941) reported experiments on traumatic shock which they interpreted as indicating the effects of a toxic factor absorbed from the traumatized region.

This view is in agreement with an enormous mass of clinical and experimental data. It is based upon sound physiological principles and it is supported by necropsy findings. The pattern of circulatory changes seen in the viscera after secondary shock is of unmistakable significance. These changes indicate the origin and nature of the circulatory deficiency. The recognition of a toxaemic factor in secondary shock will abolish the most important cause for disagreement. Investigations on other features can then go forward in an atmosphere cleared of controversial discussions.

The basic principles on which agreement may be expected are as follows: Surgical shock, like that resulting from extensive trauma, is not due to a single cause but to a combination of causes: the anaesthetic, the local loss of blood and fluid, emotional reactions, infection, or intoxication which may have reduced the patient's physiological state; the disease itself, which necessitated operation; and the absorption of toxic products from traumatized tissues. The relative importance of these factors varies in each case, and they operate in varying combinations. Some of these factors are lacking in shock from other causes.

It is now recognized that the occurrence of shock is not limited to traumatic injuries, burns, and the aftermath of extensive surgery. I have emphasized repeatedly that the same mechanisms operate in severe infections, metabolic intoxications, abdominal emergencies such as intestinal strangulation, mesenteric thrombosis, perforation, peritonitis, and from the effects of poisons.

Renal Dysfunction

Deficiency of renal function is an item of importance in secondary shock from diverse causes. For example, extensive burns are followed promptly by oliguria or anuria, by a progressive accumulation of nitrogenous wastes in the blood, and by casts, red cells, pigment, debris, albumin, and other abnormal findings in the urine. This condition persists so long as the state of shock continues, and renal deficiency becomes prominent when patients survive for several days in a state of sublethal shock (Moon, 1942a). The excretion is inadequate, the non-protein nitrogen rises progressively, hyperazotaemia develops, and the clinical syndrome of uraemia often precedes death. Similar renal disturbance occurs in secondary shock from trauma, after extensive operative procedures, intestinal obstruction, metabolic intoxication as in diabetes, icterus gravis, toxaemia complicating pregnancy, anaphylactic reactions, transfusion with incompatible blood, poisoning with various drugs, and in severe infections.

An instance of sublethal shock leading to azotaemia is that of the crush syndrome which Bywaters (1942) and others have reported. Victims of air raids had been pinioned under masonry and debris for hours. When rescued they developed signs of shock, which were counteracted by modern treatment. Despite the fact that adequate systemic circulation was maintained, these cases developed the complete syndrome of uraemia, and many died from progressive renal deficiency. When recovery occurred oliguria gave place to polyuria rather suddenly, renal function was resumed, and the non-protein nitrogen declined to a normal level. The findings at necropsy in some instances included congestion and oedema of the lungs and other viscera, as seen in secondary shock; in other cases the visceral appearances were not described. The traumatized muscles were pale, friable, and necrotic. The kidneys were pale, swollen, streaked with red and brown. The convoluted tubules showed varying degrees of degeneration, necrosis, and desquamation. The lumina contained hyaline casts, debris, and masses of brown pigment. This was shown spectroscopically to be myoglobin: its source as derived from damaged muscle is prima facie evidence of absorption from the traumatized areas.

This type of renal disorder was known to Osler and his contemporaries as "acute parenchymatous nephritis." They recognized its occurrence under several of the conditions previously mentioned. The syndrome is usually discussed as "extrarenal uraemia" by clinicians. Its pathology is not adequately presented in treatises on renal disease. Some describe it under "tubular disease," others under "nephrosis."

The exact nature and pathogenesis of renal failure accompanied by parenchymatous degeneration and necrosis in otherwise normal kidneys, as a complication of sublethal shock, is a subject for future investigations. There is suggestive evidence that the same injurious agents, including toxins and anoxia, which cause circulatory failure by their effects upon endothelium, likewise cause renal failure by their effects upon the parenchyma of the kidneys.

Practical Considerations

"The best treatment for shock is prevention." To accomplish this it is essential to know the various conditions under which shock may develop. Hence not only after trauma, extensive surgery, or burns, but also in severe infections and intoxications, and in abdominal emergencies such as thrombosis, strangulation, perforation, and the like, the anticipation of shock may make its prevention possible.

Early amputation of mangled limbs, débridement of lacerated wounds, prompt local treatment of burns as by coagulation, surgical relief of an infarcted or strangulated loop of bowel, and the drainage of infected areas are procedures which may forestall the development of shock. Likewise the prevention of infection, by therapeutic or other means, removes a potent contributory factor. The careful surgeon reduces the danger of shock by reducing to the minimum both the depth and the duration of anaesthesia and by using regional or local anaesthesia when possible. General anaesthesia usually will not of itself cause shock, but it often leaves a narrow margin of safety when combined with other shock-producing conditions.

The successful management of secondary shock requires primarily that the conditions which caused it shall be obviated or removed; otherwise, efforts to restore the failing circulation are predestined to failure. For example, unlimited quantities of plasma probably would not prevent death from intestinal strangulation or from a crushed and mangled limb unless the causative condition could be obviated effectively.

Traumatic Shock

The clinical signs of shock often develop after injuries of various kinds and degrees. Weakness, pallor, circulatory deficiency, rapid pulse, and low blood pressure are prominent features, but unfortunately these do not aid in determining either the type of the circulatory disturbance or the mechanism by which it developed. Those in whom these features are marked, apparently indicating severe circulatory failure, often recover promptly under appropriate treatment while others whose apparent condition is not alarming—they may even be ambulatory—may develop circulatory deficiency which progresses to a fatal termination despite all counteractive measures.

Experiences of this kind are well known to those dealing with accidental injuries. They have been noted especially among civilian casualties from air raids. Much confusion arises from the fact that many surgeons regard so-called *traumatic shock* as a disease entity. In reality it is nothing more than a symptom-complex, which is not a dependable index either of the *nature* of the disturbance or of its *severity*. When this syndrome is seen after injuries it is essential to differentiate between three conditions which may be operating singly or in combination. These are:

1. Primary or neurogenic shock, which may appear extremely grave and yet lead to prompt recovery. If the victim is seen within an hour or two after the injury, if the character and location of the wounds do not indicate considerable haemorrhage, and if examination of the blood does not show notable dilution or concentration, it is safe to assume that primary shock is the major factor. Any wounds should receive appropriate care, and enough morphine should be given to allay pain and apprehension. After rest, warmth, and the ingestion of fluids, recovery from primary shock is usually prompt. The experiences of surgeons indicate that anaesthesia and operative procedures should be postponed, except emergency measures, until the patient reacts from the depressed circulation. In World War I special "shock teams" were employed in casualty clearing stations and field hospitals. They supplied dry clothing and administered stimulants, heat, warm drinks, and liquid nourishment. These measures were effective in the relief of primary shock and in preparing the wounded for operation.

2. When loss of blood has been considerable the clinical signs will be like those in other forms of circulatory failure. If the person is seen within a few hours after the injury these signs probably represent the combined effects of primary shock and of haemorrhage. Examination of the blood may show which of these is the major factor. Haemodilution occurs promptly after haemorrhages; the degree of this is an approximate index of the amount of blood lost. Reports on large numbers of accidental injuries and casualties from air raids show that haemodilution is usually present. The conclusion that haemorrhage is the major factor in such cases is not open to question. The local treatment of the wounds and the restoration of blood volume, preferably by transfusion of whole blood, do not require consideration here. Cases of primary shock plus haemorrhage, if not accompanied by extensive trauma, may show marked circulatory deficiency clinically, but they usually respond readily to treatment. A tendency to progress towards a fatal termination despite treatment is not a characteristic of such cases.

3. Delayed or secondary shock results from factors other than those which cause either primary or haemorrhagic shock. The fact that their clinical signs are the same is the chief source of confusion regarding shock following trauma. This condition requires time for development: except with cranial or abdominal injuries or extensive burns, it is seldom seen until 4 or 5 hours after the injury. The chief cause for secondary shock is the absorption of toxic substances from damaged tissues. These substances originate in part from autolysis, in part from bacterial growth.

All accidental and battle wounds are grossly contaminated; bacteria, both pathogenic and saprophytic, multiply readily in the debris of damaged tissues. Probably the products of bacterial growth are more toxic than those of simple tissue autolysis. For example, shock developed more slowly and in less degree in crush injuries which were not accompanied by open wounds. When a similar amount of muscle tissue has been damaged and laid open, as by a shell fragment, the development of shock is more rapid and severe. Perhaps this is because products of bacterial growth are important factors in the latter instance.

It has been observed (Cope, 1928, 1935; Blalock, 1940) that a fall in blood pressure is not an adequate criterion for incipient secondary shock. Frequently, circulatory deficiency is developing while the blood pressure is well maintained or is even at its highest recorded point. So long as the mechanism of compensation is effective there is no marked decline of the arterial pressure; the latter indicates that the mechanism of compensation is failing and that the condition is becoming critical. An ominous fall in blood pressure is not a sign of *incipient* secondary shock; too often it is a sign of *departed opportunity*.

As stated earlier in this paper, secondary shock is accompanied by a disturbance of fluid balance which results from abnormal endothelial permeability. This disturbance affects the mechanism of absorption. Fluids given orally or subcutaneously are not absorbed. Thirst becomes incessant, but efforts to relieve it are followed by vomiting. The fluid vomited is often in excess of that swallowed, and the vomitus in this condition contains brown flecks—"coffee grounds" vomitus. These flecks contain haemoglobin; they result from capillary haemorrhages in the gastric mucosa.

Haemoconcentration

Abnormal endothelial permeability which disturbs fluid balance results in haemoconcentration—a distinguishing feature in this type of shock. This feature is valuable both in recognizing the type of circulatory deficiency and in noting its progress. Haemoconcentration may be shown by counts of erythrocytes, by haemoglobin readings, by haematocrit, or by the specific gravity of the whole blood. My personal preference is for erythrocyte counts. Whichever method is used, a reading should be made when the injured person is first seen. Subsequent readings at intervals of one to three hours will furnish valuable evidence as to the nature and progress of the condition.

Unfortunately haemoconcentration may be masked by the haemodilution resulting from associated haemorrhages. Also, the victim may have had some degree of anaemia before the injury. For example, an erythrocyte count of 5,000,000 appears normal, but in one whose previous count was 3,500,000 it would indicate more than 40% haemoconcentration.

If the first examination shows moderate haemodilution it is apparent that haemorrhage is the major factor; subsequent findings of the same degree or of lower dilution would likewise indicate haemorrhage. But in the same case a subsequent finding of a *higher* concentration some hours after the injury indicates that secondary shock is developing in one previously affected by haemorrhage or perhaps by anaemia. If a person seen for the first time four hours or longer after injury shows clinical signs of shock *not accompanied by haemodilution*, it suggests that neither primary shock nor haemorrhage is the cause. Inadequate circulation developing some hours after injury, with approximately a normal concentration of the blood, is strongly presumptive of secondary shock in one who has lost blood by haemorrhage, or perhaps in a previously anaemic person.

The incipient stage of secondary shock unaccompanied by haemorrhage may be detected by the presence of moderate haemoconcentration—10 to 20%—even before evidence of circulatory deficiency is manifested clinically. This is seen frequently within an hour or two after extensive burns because shock develops faster after burns than after other injuries. If a subsequent examination of the blood shows a higher degree of concentration it indicates progress in the development of shock even though the arterial pressure may not be ominously low.

In order to treat the patient intelligently it is of advantage to know which condition or combination of conditions is responsible for the circulatory deficiency. A consideration of the time factor plus observations on the concentration of the blood may be distinctly helpful.

Treatment

Several therapeutic misconceptions are prevalent concerning the treatment of secondary shock. There is no apparent basis for the belief that morphine is useful in counteraction or prevention. On the contrary, morphine is dangerous when given in full doses because it inhibits respiration and thereby increases anoxia, which is a major factor in this condition. Enough morphine should be given to allay pain and apprehension, but amounts sufficient to lower the respiratory rate will accelerate the development of secondary shock.

Experience has shown that stimulants, such as digitalis, strychnine, and adrenaline, not only are useless but may be distinctly harmful. Cardiac deficiency is not a factor in the development of secondary shock; the vasomotor system is active and the peripheral arteries are already contracted to the maximum. Under such conditions adrenaline cannot be expected to produce benefit, and it should be remembered that large or repeated doses will actually cause shock in experimental animals.

Another matter deserving thoughtful consideration is the application of heat in attempts to prevent or to counteract shock. It is natural that physicians should note the low peripheral circulation and temperature and should try to relieve them artificially. The application of heat is widely practised, perhaps without due consideration of the physiological principles involved. During shock and after haemorrhage the blood volume is reduced. There is not sufficient blood to fill the entire vascular bed and to support metabolism throughout the body. Physiological reactions compensate for this condition by a selective redistribution of blood, favouring vital organs at the expense of the external parts. The vascular channels in non-vital areas become collapsed and bloodless, but both circulation and metabolism are maintained where they are most needed.

It is possible to open up the peripheral vascular bed by the application of heat and stimulants. But this counteracts Nature's mechanism for conservation: the blood required to restore circulation in the external areas lowers needlessly the already depleted reserve of blood. Recent evidence, both experimental and clinical, indicates that the use of heat in the treatment of shock causes further circulatory deficiency and that the results are detrimental.

Those remedies which have proved useful are agents which tend to counteract one or another of the major items by which secondary shock develops or progresses; chief among these items are *anoxia*, *endothelial permeability*, and *reduced blood volume*. Lack of oxygen accelerates the development and progress of shock. The inhalation of oxygen aids in counteracting this and in preventing irreparable damage. It raises metabolic activity and tends to interrupt the action of the vicious circle even after shock has been initiated.

Physiological studies indicate that one function of the adrenal cortical hormone is to maintain the normal impermeability of endothelium. There is significant evidence, both experimental and clinical, that this substance is distinctly beneficial in the prevention and in the treatment of shock from burns (Wilson *et al.*, 1936; Rhoads *et al.*, 1941).

The successful treatment of shock requires, first, that the condition which caused it shall be removed; next, that the fluid which was lost from the blood be replaced by fluid which should approximate closely the composition of that lost. Thus after serious haemorrhages transfusions of whole blood provide the ideal remedy. But in shock accompanied by haemoconcentration whole blood is not so suitable; it does not relieve the haemoconcentration, which, because of viscosity, tends to impede circulation through the capillaries. The patient does not lack erythrocytes, but he sorely needs fluid to restore the blood to its normal composition and volume. These considerations indicate clearly that the ideal replacement fluid is either human plasma or serum.

These substances are now available in most hospitals and medical centres, either in the original form or concentrated or dehydrated. Plasma or serum, given in the normal or isotonic concentration, serves admirably as a replacement fluid to restore blood volume and to reduce haemoconcentration. But the same amount of plasma, concentrated to one-half or even to one-fifth of its original volume, is far more effective in counteracting both the low blood volume and the deranged equilibrium of fluids which are prominent factors in the mechanism by which shock develops (Hill and Muirhead, 1942).

Concentrated plasma or serum, given intravenously, raises the osmotic pressure of the blood and thereby draws fluid from the tissues into the vascular system. This accomplishes several desirable results: it increases directly the total blood volume; it relieves the haemoconcentration, reduces the viscosity of the blood, and restores fluid balance; it tends to reduce the oedema of soft tissues; it also relieves the hypoproteinaemia which often is a notable feature. These effects increase the circulatory efficiency and the delivery of oxygen to the tissues. The relief of anoxia removes a most important factor from the vicious circle by which the deficiency progresses.

The foregoing considerations are not based upon personal experiences. They represent an analysis of the mechanisms involved and a comparison of these with the recorded experiences of physicians and surgeons.

Let it be emphasized that *early* recognition and treatment are of the highest importance if favourable results are to be hoped for. Treatment, to be effective, must anticipate the complicated interplay of the agencies whose combined effects culminate in the syndrome of shock. It must be administered before the failing circulation and the associated anoxia have produced irreversible changes in tissues whose functions are vital.

Summary

Confusion arises from the fact that three unrelated mechanisms may cause the clinical signs of shock following injuries. These are: primary or neurogenic shock, the effects of haemorrhage, and delayed or secondary shock. These may operate singly or in combination.

Traumatic shock is not a disease entity but a syndrome. It represents the summative effects of the three conditions mentioned. Each of these causes low arterial pressure, hence they cannot be distinguished by that criterion.

The physiological disturbances which accompany secondary shock are not the same as those of haemorrhage. Simple uncomplicated haemorrhages apparently will not produce this syndrome. But haemorrhage, when present, is a highly important contributory factor: a person in whom secondary shock is developing may not withstand the loss of an amount of blood which would be insignificant in an otherwise normal person.

Loss of capillary tonus and abnormal permeability of endothelium are important factors in *secondary* shock. These have not been shown to play a part either in primary shock or in the effects of haemorrhage. Abnormal permeability disturbs the mechanism of fluid balance, leading to oedema of the viscera and to haemoconcentration.

A serious derangement of renal function accompanies secondary shock. This is seen not only after injuries and burns but also from the effects of intestinal obstruction, mesenteric thrombosis, metabolic intoxications, anaphylaxis, transfusion reactions, severe infections, and from the effects of various poisons. Patients who survive several days in a state of sublethal shock frequently develop the syndrome of uraemia and die of renal dysfunction.

The clinical management of shock requires the recognition of the causative mechanism. Cases of primary or haemorrhagic shock, unless accompanied by extensive trauma, respond more readily to treatment than do those of advanced secondary shock.

Observations on the concentration of the blood and a consideration of the elapsed time since the injury are valuable aids in determining which mechanism is the major factor causing circulatory deficiency.

Loss of blood volume is an outstanding feature after haemorrhages and in secondary shock. The one is best treated by transfusion of whole blood; the other by plasma or serum.

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MENINGITIS DUE TO PITTMAN AND NON-PITTMAN STRAINS OF H. INFLUENZAE

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For the last few years treatment with sulphonamides has been extensively applied to cases of meningitis due to *H. influenzae*. In this country several cases treated successfully in this way have been recorded: Jones (1937), Teggart (1938), Roberts (1939), Sakula (1940), Harold (1941), Jacoby (1941), Archer and Singer (1943), Birch (1943), and Matthews (1943). None of these reports give any clue as to the serological type of the *H. influenzae* strains isolated. Two reports have been published in this country about unsuccessful treatment with sulphonamides of meningitis due to *H. influenzae*—Mackenzie, Page, and Ward (1940) and Jones and Sudds (1942). The latter paper is the only one in which a serological examination of the organism responsible was carried out; the culture was of the b type (Pittman, 1931).

A third group of papers give accounts of both successes and failures in *H. influenzae* meningitis (serological types not given) using the sulphonamides for treatment: Mutch (1941), Davies (1943), and Moir (1943). Davies and Moir, however, emphasize the importance of using large doses of these drugs at an early stage. Moir also stresses their continued use after meningeal symptoms have disappeared, in order to avoid relapses. Mutch has suggested that strains isolated in this country should be examined not only culturally but also serologically. Mutch and Davies are not very favourably impressed by the curative value of these drugs in this condition, particularly with infants.

American Views on Treatment

American publications on this subject are more numerous, and several reviews have been published: Bigler and Haralambie (1939), Aléman (1940), Neal, Appelbaum, and Jackson (1940), Lindsay, Rice, and Selinger (1940), Alexander (1941), Mitchell (1941), Neter (1942), Alexander, Ellis, and Leidy (1942), and Knouf, Mitchell, and Hamilton (1942). The general tone of the reviews is that sulphapyridine is more useful than sulphanilamide in the treatment of these cases, and some authors claim that the combination of an antiserum to *H. influenzae* and sulphapyridine provides the best form of treatment available at present. Neal, Appelbaum, and Jackson (1940) reported on 29 patients, 20 of whom were treated with this combination, 9 being given sulphapyridine alone. Of the 29 patients 14 recovered. The mortality rate was therefore 52%. Of the 14 patients recovered 5 had received the drug alone. Lindsay, Rice, and Selinger (1940), in reviewing 108 cases, showed that treatment with antisera reduced the mortality from 97% to 83%: by adding sulphanilamide and sulphapyridine the mortality for 13 cases was further reduced to 54%. Knouf, Mitchell, and Hamilton (1942) report altogether on four comparable series of 63 patients treated during the last 10 years. Group 1 consisted of 19 patients treated only symptomatically. In this group all patients died. Group 2 also had 19 patients. These were treated with anti-influenza serum. All died. In Group 3, which consisted of 13 sulphanilamide-treated patients, there was one survivor. Group 4 was made up of 12 patients, with 9 survivors, or a mortality of 25%. The patients in this group were treated with sulphapyridine.

Strains of H. influenzae

Pittman (1931) had shown that the majority of *H. influenzae* strains isolated from meningitis cases in the United States belonged to a special group of this organism, which is characterized by possessing a capsule consisting of carbohydrates. This capsule has antigenic properties. Six different types of capsular antigens can be differentiated, and the American meningitis strains belong mostly to Type b. According to Pittman capsulated strains have a smooth appearance, but the colonies are rough when the strains lose their capsules, whereas the strains usually isolated from the respiratory tract are non-type-specific and have a rough appearance when first isolated. Fothergill and Chandler (1936), on the strength of virulence and agglutination experiments on dissociated smooth strains, regarded all rough strains as avirulent variants of *H. influenzae*. Pittman (1939) could protect mice easily with sulphapyridine against infection with *H. influenzae* (non-type-specific). Pittman (1942), using *H. influenzae* (Type b) and confirming the findings of Povitzky (1937) with meningeal strains, showed that in experimental infections of mice the combination of antiserum and sulphapyridine gave a higher percentage of cures than either of these substances alone. Pittman (1942) also found sulphanilamide not very effective as compared with sulphapyridine, sulphathiazole, and sulphadiazine; furthermore, that several strains of *H. influenzae* (Type b) showed different sensitivity to the drugs. There are two fatal meningitis cases on record (Pittman, 1933; Mulder, 1939) in which rough strains (non-type-specific) were isolated from the cerebrospinal fluid. Neither of these two cases was treated with sulphanilamide or any of its compounds. Pittman (1931) has shown that rough strains, and Allison, Gordon, and Zinnemann (1943) have shown that respiratory strains not belonging to the Pittman types, are virulent for white mice, although this virulence is lower than that of strains of the Pittman types. In view, therefore, of the small number of cases reported as being due to non-capsulated strains of *H. influenzae*, and their theoretical importance, we are recording the following cases of meningitis, which responded very well to sulphapyridine and which could be shown not to belong to any of the Pittman types.

Non-Pittman Type of Meningitis treated with Sulphapyridine

Case 1.—G. R., aged 7. Fell from tricycle Aug. 14, 1942. Persistent headache and vomiting since. On admission, Aug. 21, T. 97.6°, P. 92, R. 20; appeared fairly well; quiet. Pupils equal, reacted briskly. Some neck rigidity. Abdominal reflexes equal and active. Knee-jerks—R. normal, L. almost absent. Triceps jerks equal. No Kernig, no Brudzinski, no Babinski. Tone of limbs normal. Swelling size of a walnut L. occipital region. Lumbar puncture: 10 c.cm. fluid removed; no increase of pressure; fluid slightly turbid. Aug. 22: Mentally very bright; no pain; neck rigidity marked; Brudzinski positive; Kernig negative, but test causes obvious pain; knee-jerks not obtained; T. 100°, P. 114, R. 22. Aug. 23: Neck rigidity less; T. normal, and never raised again. Aug. 24: Cyanosis (from sulphapyridine). Aug. 25: Neck rigidity almost gone. Aug. 27: No neck rigidity; no knee-jerks, otherwise appeared normal. Sept. 5: Discharged quite well. *Treatment:*—Aug. 21: Sulphapyridine 2 g. (4 tablets) stat.; 1.5 g. four-hourly for 24 hours; alkaline mixture (pot. cit. gr. 40, sod. bic. gr. 20) given four-hourly along with sulphapyridine. Aug. 22: Sulphapyridine 1 g. four-hourly. Aug. 24: Sulphapyridine 0.5 g. four-hourly. Aug. 26: Sulphapyridine discontinued. Total dose of sulphapyridine, 26 g.

Case 2.—T. S., aged 5. Admitted March 3, 1943. Since March 1 had had headache and vomiting. On admission did not seem ill;

T. 102.4°—after that it was never above normal. The only abnormal signs were head retraction, neck rigidity, and positive Kernig. A lumbar puncture was performed on admission, and 25 c.cm. of turbid fluid removed under pressure. He was given sulphapyridine 2 g. on admission, and over a period of 7 days a total of 21 g. On March 5 he appeared much brighter and the neck rigidity was less marked. On March 9 he seemed quite well.

The colonies of the strains of *H. influenzae* isolated from Cases 1 and 2 had serrated edges when first isolated, and were in no way found to differ from the strains which we regularly isolated from cases of bronchiectasis. Strain 1 (G. R.) gave a negative and Strain 2 (T. S.) a positive indole reaction.

Agglutinations were carried out with the technique described by Allison, Gordon, and Zinnemann (1943). Neither strain gave a specific agglutination with the agglutinating *H. influenzae* sera a to f when incubation was carried out at 37° C. An agglutinating serum, prepared by immunizing a rabbit with the strain isolated from Case 1, did not agglutinate the Pittman strains a to f at 37° C., nor did the serum agglutinate the strain from Case 2, but it gave a good agglutination with its own strain. Agglutinations were also carried out at 56° C. to exclude the possibility of the strains' being rough variants of any of the Pittman types. According to Pittman (1931) and Mulder (1939), agglutinations at higher temperatures are not due to type-specific but to species-specific antibodies.

Strain 1 gave a marked agglutination at 56° C. with the serum prepared from this same strain, whereas Strain 2 did not give a specific agglutination with this serum. None of the Pittman strains a to f gave a specific agglutination with the serum produced with Strain 1. Neither Strain 1 nor Strain 2 gave a specific agglutination with any of the Pittman agglutinating sera a to f at 56° C. We therefore believe that the strains of *H. influenzae* isolated from these two cases of meningitis are not different from strains commonly isolated from respiratory conditions.

Although such strains have a lower virulence than capsulated *H. influenzae* strains, they are by no means avirulent. The virulence of these two strains was tested by injecting 0.5 and 1 c.cm. of 24-hours Levinthal broth cultures intraperitoneally into 6 mice—3 mice for each dose. The mice injected with 1 c.cm. of culture died within 5 days and a culture of *H. influenzae* was recovered from the heart blood. Of the mice injected with 0.5 c.cm. of culture, none of those injected with Strain 1 died, and only one out of the three injected with Strain 2 died. This is in contrast to the virulence of the meningeal type Pittman b, which is much more virulent to mice, and of which 0.5 c.cm. injected intraperitoneally kills constantly within three days all the mice injected.

H. influenzae strains possessing the characteristics of respiratory strains are therefore apparently able to cause meningitic conditions in man.

As is evident from the literature quoted in the early part of this paper, very few cases of *H. influenzae* meningitis have been typed serologically in this country. We are therefore giving an account of the following three cases which occurred recently after the second case described. The strains isolated from these cases were of the Pittman type b.

Pittman Type b Cases

Case 3.—H. D., aged 5. This boy was admitted to the Brotherton Wing of the General Infirmary at Leeds under the care of Dr. Wilfred C. Vining, to whom we are indebted for the clinical notes of this case. On May 26, 1943, the child complained of headache and loss of appetite, and became irritable and restless. He was first seen on May 29. The condition was then semicoma. There were stiffness of the neck and incontinence, and the knee-jerks were absent. He was admitted the same afternoon. Sulphathiazole was administered —4 g. to be given in the first 24 hours ; and intramuscular sulphapyridine 1 g. immediately. Lumbar puncture. Fluid was under considerable pressure and grossly turbid. Cells: 655 polymorphs per c.mm. The patient died within a few hours of admission, his illness lasting only about 3 days. The film from the centrifuged deposit of the c.s.f. showed masses of pleomorphic Gram-negative bacilli and numerous pus cells. On culturing, a profuse growth of *H. influenzae* was obtained. The colonies had a moist opaque appearance on chocolate agar, characteristic of the capsulated Pittman types, and on agglutination with the various type antisera gave a marked specific agglutination with Type b antiserum. Wet preparations from the agglutinated culture stained with methylene blue showed swelling of the capsules.

Case 4.—I. A., aged 2. A portion of brain from this girl was received from an inquest, at which it was stated that the child had not been ill except on the day before death. At the necropsy an acute endocarditis and a meningitis with only a small exudate were found. A pure culture of *H. influenzae* was obtained from the meningeal exudate. The colonies were opaque and of a smooth mucoid appearance. The strain gave a marked agglutination with Type b antiserum. Swelling of the capsules could be seen in wet preparations.

As these two cases had such a fulminating and fatal course, and as treatment was begun only a few hours before death occurred in the one case and no treatment at all was administered in the other, they cannot be compared from the point of view of therapy with the other two cases. The third case in this series was the following:

Case 5.—V. S., aged 5. This girl was admitted on Nov. 1, 1943, to the Brotherton Wing of the General Infirmary at Leeds under the care of Dr. J. R. H. Towers, to whom we should like to express our thanks for the clinical notes of the case. She had started three days previously with general malaise and pyrexia followed by vomiting. Apparently the evidence of an intracranial lesion appeared at the end of the second day, with severe headache, some neck retraction, and confusion. These were followed by gradually deepening stupor. At the time of admission marked head retraction and Kernig's sign were present, with a temperature of 102°. Lumbar puncture revealed turbid cerebrospinal fluid, which contained 2,880 polymorph leucocytes per c.mm. and Gram-negative filamentous rods, which on subculture proved to be *H. influenzae*. This strain gave a characteristic agglutination with the *H. influenzae* Type b antiserum, and swelling of the capsules was seen in wet preparations. Treatment was with sulphapyridine, 2 g. being given first intramuscularly, then 1 g. four-hourly by mouth, and later 0.5 g. four-hourly. There were three recurrences of temperature, each accompanied by headache and each one following the cessation of sulphapyridine therapy. The cerebrospinal fluid withdrawn after the first recurrence (Nov. 6) contained about 16 cells per c.mm., and on culture gave a scanty growth of *H. influenzae*, serologically of Type b. The cell content on the second recurrence (Nov. 18) rose to 164 per c.mm. The total amount of sulphapyridine given was 46.25 g. The patient was discharged on Dec. 9, symptom-free, and has remained well since.

This fifth case responded to sulphapyridine treatment, though not as easily as Cases 1 and 2, and illustrates the advisability of continuing treatment in cases of *H. influenzae* (Type b) meningitis for some time after the temperature has returned to normal and bacteriological examination of the cerebrospinal fluid has established the absence of *H. influenzae*.

There does not seem to be much known at present regarding the relative response of non-type-specific and type-specific strains of *H. influenzae* to sulphapyridine or any other sulphonamide. We think, therefore, that it would be advisable, wherever possible, to type all strains of *H. influenzae* from cases of meningitis with a view ultimately to determining any differences in prognosis and treatment of these cases.

Summary

Two cases of meningitis caused by *H. influenzae*, and their successful treatment with sulphapyridine, are described.

According to agglutination reactions, the strains isolated from these cases do not belong to any of the six Pittman *H. influenzae* types ; apparently they are not different from the strains commonly isolated from respiratory conditions. Previously only two cases caused by such strains have been described.

Two fulminant and fatal cases which were found to be due to *H. influenzae* Type b are recorded. Hardly any treatment could be given in one case and no treatment at all in the other ; these cases therefore cannot be compared with the other two cases from the point of view of therapy.

A clinically severe case of meningitis due to *H. influenzae* Type b is described, which responded well to sulphapyridine treatment. The three recurrences of meningitic symptoms after cessation of sulphapyridine suggest a continuation of therapy for a time after the return of the temperature to normal and after the cerebrospinal fluid has proved to be free of *H. influenzae*.

It would be advisable, in our opinion, to type as many strains as possible from cases of meningitis due to *H. influenzae*

with a view to determining any possible differences in prognosis and treatment.

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SULPHONAMIDE ALLERGY

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Sensitization to sulphonamide drugs may arise after internal or external administration. In the latter case the reaction is an eczematous dermatitis, and in the former a syndrome consisting of drug fever, skin eruption, leucocytosis, and—more uncommonly—joint effusions, conjunctivitis, jaundice, agranulocytosis, or other features.

When a patient develops any of these types of allergy, oral administration of the drug causes sharp reactions which render future sulphonamide therapy unpleasant or dangerous. In these cases the question which naturally arises is whether such reactions will occur if other drugs of the sulphonamide series are given. This is a point which has received little attention up till now. Moeschlin (1942) reported six cases sensitized to sulphathiazole, of which two reacted to other sulphonamides. Haviland and Long (1940) exposed two sulphathiazole-sensitive patients to sulphapyridine and obtained reactions. Cases of both single and multiple sensitivities have been described by Erskine (1938), Goodman and Arthur (1941), Bloom (1942), Costello (1943), and Swartz and Lever (1943).

All these cases followed internal administration. That the eczematous form of allergy can also exist to single or multiple sulphonamides was shown in a previous report (Park and Platts, 1942). The investigation reported here was undertaken to determine the frequency of these two types of allergy.

Methods Used

Forty cases of both internal and eczematous allergy have been investigated by skin tests and oral medication with various sulphonamides and the related compounds, sulphanilic acid and procaine.

For oral testing, a mixture was used which disguised the slightly bitter taste of the sulphonamide and in which different drugs could be given without the patient being aware of any change. The first dose was usually 0.5 g., and was worked up in the absence of a reaction to 1 g. four-hourly for 48 hours. Reactions in the eczematous cases consisted of a local flare-up plus a general dermatitis, starting in about six hours, and, in the internal cases, of pyrexia starting in about two hours.

Skin tests were used in the eczematous cases only. Since ordinary patch tests are unreliable in sulphonamide eczema, a modified scratch technique was used, with 50% pastes of powdered sulphonamides in hydrous lanolin and a 2% lanolin paste of procaine. These were either rubbed repeatedly into the scratch or applied over it as a patch. The final method evolved was to apply four different test materials to one long scratch on pieces of cloth about an inch apart, and cover with adhesive for 48 hours. The positive reaction was seen on that part of the scratch in contact with the offending substance only.

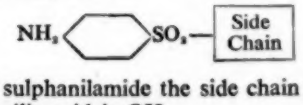
Results

The results of the tests are shown in the following table:

Reaction	Sulphanilamide		Sulphapyridine		Sulphaguanidine		S'thiazole	Others
	Skin Test	Oral	Skin Test	Oral	Skin Test	Oral	Oral	
Eczema ..	+	+		+		+		
" ..	+	+		0		0	0	
" ..	+	+		?		0	0	
" ..	+	+	+	+		+	+	Procaine skin test +
" ..		+				0	0	
" ..		+		+				
Fever, rash, neutropenia		0		+				
Eczema ..	+				0			
" ..	+	+			+			Procaine 0
" ..	+	+	0		0			
" ..	+	+	0	0	0			
" ..	+				+	+		Procaine +
" ..	+	+		0	0			
" ..	+	+			0			Procaine 0
" ..	+	+	0		0			
" ..	+	+		0	0			
" ..	+	+		0				
" ..	+	+		0	0			
" ..	+	+		0				
" ..	+	+		+	+			Sulphanilic acid + ; proc. +
" ..	+	+		0	0			
" ..	+	+		+	+			Procaine 0
Fever, rash ..		0		+				
Eczema ..	+			+	+			S. acid + proc. 0
Photosensitivity		+		0				
Eczema ..	+				+			S. acid and proc. +
" ..	+	+	+		+			Procaine +
" ..	+		0	+	?			S. acid and proc. +
" ..	+		0		0			S. acid and proc. 0
Fever, rash ..		+		0			0	S. acid 0
" " ..		+		+				
Eczema ..	+	+	+		+			Procaine +
" ..	+	+	+		+		+	S. acid + ; proc. 0
" ..	+	+	0	0	0			
Fever, rash ..		+		0			0	S. acid 0
" oedema		+		+			0	S. acid 0
Eczema ..	+	+	+		+			S. acid + ; proc. 0
Fever, rash ..		+		+			+	S. acid +
Eczema ..	+	+	0	0	0		0	
Fever, rash ..		0					0	Sulphadiazine 0
								Sulphamezathine +

Discussion

All drugs of the sulphonamide series consist chemically of the para-amino-phenyl-sulphonic radical ($NH_2.C_6H_4.SO_2$–), linked to various side chains, thus:



In the case of sulphanilamide the side chain is NH_2, and in the case of sulphanilic acid is OH.

Of these 40 cases 24 reacted only to the individual drug by which they were initially sensitized, thus being allergic to the sulphonamide group plus all or part of the side chain. The last case (see table) shows how highly specific this allergy can be, being insensitive to sulphadiazine but sensitive to the same compound with two methyl groups added (sulphamezathine).

Allergy to Multiple Sulphonamides.—It was found that no case was allergic to more than one of the series without being allergic to them all and to sulphanilic acid. The group common to all

these is the sulphonamide or para-amino-phenyl-sulphonic radical, $NH_2.C_6H_4.SO_2-$. Of the 40 cases 16 were of this type. Two cases not included in this group, however, behaved in a peculiar manner. They were both eczemas, highly sensitive to small doses of sulphanilamide but not to small doses of other sulphonamides. Each of them reacted to other sulphonamides in *heavy* dosage, with mild eruptions only. This reaction included a selective flare-up of the sites of previously positive sulphanilamide skin tests, but not of the negative tests to other sulphonamides. Had they been allergic to the sulphonamide group they should have reacted to all these in roughly equal amounts (1 g. of sulphanilamide or sulphanilic acid is equivalent to about 1.5 g. of sulphapyridine or sulphathiazole). The marked difference suggests that a small quantity of sulphanilamide or closely related compound may be formed in the body from the other sulphonamide drugs.

Allergy to Other Drugs.—Rogers (1938) described a case in which the administration of sulphanilamide caused a flare-up of the sites of several previous reactions to procaine. Procaine has the formula $NH_2.C_6H_4.COO.CH_2.CH_2.N(C_2H_5)_2$. It therefore has in common with the sulphonamides the amino-phenyl radical, $NH_2.C_6H_4-$. Skin tests with procaine were made on 14 of the above cases allergic to multiple sulphonamides. Seven gave positive reactions. Four of these were tested with full oral doses of phenazone, which carries the radical $NH.C_6H_4-$, but none gave any reaction.

A possible case of sensitivity common to both sulphathiazole and arsphenamine, which also contains the amino-phenyl radical, has been reported by Weinstein and Domm (1941).

Summary

The specificity of the sensitivity in 40 cases allergic to sulphonamide drugs has been investigated.

In 60% the allergy was confined to one sulphonamide drug. In 40% it occurred to multiple sulphonamides and to sulphanilic acid.

In about half of the latter allergy was to the sulphonamide radical, $NH_2.C_6H_4.SO_2-$. In the other half it was to the amino-phenyl radical, $NH_2.C_6H_4-$, reactions also occurring to procaine.

No cases were seen in which the sensitization was to still smaller groups than these.

I wish to thank the Commanding Officer, 1 N.Z. General Hospital, and the staff of the hospital, for the facilities and assistance in carrying out this work; and the Director of Medical Services, 2 N.Z.E.F., for permission to publish this report.

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TREATMENT OF PULP INFECTION OF THE FINGERS IN THE FIELD

BY

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Major, I.M.S.

The following is based on observations made after three years in casualty clearing stations, during which time a large number of these cases have been treated. The main object of treatment was to avoid evacuation to the base, and to return the patient as soon as possible to his unit.

There is little need to labour the disability caused by a stiff and painful finger, especially if it be the index finger of the right hand. The finger may be merely useless or, as often happens, an actual hindrance, interfering with the use of the whole hand. It is not proposed to discuss the aetiology at length. Septic cuts, scratches, and unnoticed pricks are probably the chief precipitating causes. Contributory are the difficulties of ensuring asepsis by reason of water shortage, dust, and the nature of the man's work; this is particularly so in the case of small parties.

Pathologically the lesion is at first a simple pulp infection, though lymphangitis, lymphadenitis, and general symptoms are not slow in supervening; untreated or inadequately treated bone necrosis follows with depressing frequency, with its consequent increase in the period of disability. The symptoms of the established case need little description. Severe and throbbing pain and complete loss of function are the salient features. General symptoms may be fairly severe, with headache and pyrexia; the finger is tense, red, and extremely tender. Here it may not be out of place to emphasize that the utmost gentleness in examination is essential. The patient will have to be re-educated in the use of the finger and hand: the more pain he suffers, the longer this is going to take.

Management of Cases in Detail

The successful management of these cases calls for attention to detail: a man may be off duty longer with a whitlow than after appendicectomy. In my experience, conservative methods are unavailing; the sulphonamide drugs are useful, but they will not cure the patient without surgical treatment. The primary stage in the treatment is the establishment of adequate relief of tension, and that as soon as possible. "Waiting for pus to form" is mentioned only to be condemned.

The operation is simplicity itself, and I have done many in field ambulance dressing stations with entirely satisfactory results. The anaesthetic of choice is pentothal sodium or some other intravenous barbiturate. If possible, this is preceded by morphine gr. 1/4 and hyoscine gr. 1/150. Adequate drainage is secured by laterally placed bilateral incisions, the pulp space is freely opened with fine Spencer Wells forceps, and a piece of glove rubber drain put across. The incision should not, and need not, traverse the flexure creases of the finger. For infections in the terminal phalangeal area it is, I think, not necessary to carry the incision in an acute case right across the tip of the finger. The drain is taken out after 48 hours; gauze and thick rubber drain are inferior to glove rubber in every way.

The incision of the whitlow is by no means the end of the case from a surgical standpoint. Morphine should, I consider, not be withheld for the first two nights, as a transient increase in the swelling may follow the incision. The incision is dressed once daily with hypertonic saline: Lieut.-Col. P. D. Johnson has suggested the use of sterile 12% sodium sulphate, and in my personal experience this is excellent. Every three hours warm sterile solution is poured over the dressing, which is undisturbed. Heat by means of a hot bottle or, if available, an electrically heated blanket is applied continuously for the first three or four days. I am not convinced that the sulphonamides greatly influence the condition, for after drainage the lymphatic and general conditions rapidly subside.

As soon as the incisions are clean, and swelling is subsiding, the encouragement of active movements should be begun. At no time should any attempt to force movement be made: it merely brings home to the patient that his finger is painful and useless. Progress is neither rapid nor spectacular. When incisions are healed the whole hand is placed in warm water and active movements persisted in. Passive movement is the worst form of interference. The man must be given to understand that further progress depends upon himself. This is the most difficult and trying part of all: it may take up to three months to secure full return of function. This time can be spent usually in the man's unit, supervised by his medical officer.

Summary

This paper on pulp infection of the fingers has been written largely from the military standpoint.

Emphasis has been laid on early, adequate, but not mutilating incisions to secure relief of tension.

Incisions are not to be made primarily to "find pus" but to prevent phalangeal necrosis by relieving pulp-space tension. Reliance should not be placed on the sulphonamide drugs as substitutes for incision.

Prolonged soaking of the finger should be avoided, and a method of avoiding this is suggested.

Active movements should be encouraged and all attempts at passive forcing should be avoided.

Extreme gentleness in handling is at all times necessary in order to avoid psychological trauma as well as physical discomfort. A case which has been dealt with at the field ambulance is more comfortably evacuated to the C.C.S. than the patient upon whom no operation has been performed. The equipment necessary is found in every field ambulance and C.C.S.

In the presence of general symptoms it is a wise precaution to take a blood film in malarial areas. It is illuminating to note how often an attack of malaria is initiated by the whitlow.

I wish to thank Lieut.-Col. P. D. Johnson, R.A.M.C., for permission to write these notes and also for help given and suggestions made.

EARLY OPERATION FOR VOLKMANN'S ISCHAEMIC CONTRACTURE

BY

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The recognition and adequate treatment of Volkmann's ischaemic contracture in its early stages will result in a good functional recovery in most cases. Failure to appreciate the significance of the early developing signs of this lesion is, unfortunately, the cause of much subsequent disability and, not seldom, of even gross crippling deformity.

In a recent standard textbook on orthopaedic surgery, in a paragraph dealing with the question of operations designed to relieve intrinsic pressure on the vessels in the developing stages of Volkmann's deformity, it is stated that "as a rule damage is already irreparable when the arm is observed, and for this reason the operation is seldom applicable." Too complete an acceptance of this ruling will result in many cases progressing to ischaemic deformity which would otherwise have been prevented had the benefits of early surgery been sought. I believe that operative exploration is indicated if arterial damage is suspected, or where gross swelling of the forearm is associated with beginning contracture of the fingers which has not responded to routine treatment. The case history given below demonstrates the good results which do follow surgery in the early stages:

Case History

While on Army manœuvres on Dec. 4, 1943, the patient, a well-developed man of 24, was hit accidentally by fragments from a ricocheting bullet. He felt an immediate burning sensation in the left forearm. A first-aid dressing was applied and the patient was admitted to an E.M.S. hospital.

When examined in the hospital four hours after the injury several small wounds were seen on the flexor aspect of the proximal third of the left forearm. There had been very little bleeding. Flexion and extension of the elbow were full and painless, but supination and pronation were reduced by 50% and were accompanied by pain. The fingers were slightly flexed, but could be extended at the metacarpo-phalangeal joints. There was no loss of sensation in the hand or forearm, and the radial pulse was easily palpable. Prophylactic doses of anti-gas-gangrene serum and antitetanic serum were given, and the administration of sulphapyridine was begun. The forearm was suspended from a Balkan frame to assist in venous return.

Three days after admission the range of movement of his fingers had increased, but there was a tendency to flex the wrist before full extension of the fingers was secured. Twelve days after admission to hospital four small pieces of metal were removed from the forearm under local anaesthesia. Following this minor procedure, the patient experienced an aggravation of pain in his arm and the degree of contracture of his fingers was found to have increased.

When seen on Dec. 24—20 days after injury—the muscles of the proximal part of the forearm were found to be hard, and the patient could not extend his fingers at the metacarpo-phalangeal joints without at the same time flexing the wrist fully. Volkmann's ischaemic contracture was diagnosed and surgical exploration decided upon. This was done through a linear incision on the volar aspect of the proximal third of the left forearm. The skin and fascia were incised, and superficial muscles which appeared normal were separated. When the insertion of the pronator radii teres was elevated and later partially incised, large blood clots welled up into the wound and profuse arterial bleeding occurred. This was found to arise from a partially lacerated ulnar artery. No arterial spasm was present in either segment. Ligatures were applied above and below the laceration and the vessels divided. The deeper muscles were soft, dark red in colour, and failed to show any sign of contractility on stimulation even when a small section was removed from the flexor digitorum profundus for microscopical study. The divided structures were reunited, the wound closed in layers, and the wrist fixed in hyperextension on a dorsal plaster cast. A small rubber drain was left in for 24 hours.

The stitches were removed at the end of 12 days, when the wound was found to be well healed. There was still, however, slight flexion contracture of the distal phalanges of the index, middle, and ring fingers. Splinting in hyperextension was reapplied and physiotherapy by infra-red rays was begun. On Jan. 19, 1944 (26 days after operation) the patient had recovered full finger and wrist movement.

I am indebted to Col. W. F. Harvey for the following histological report: "The sections showed muscle fibres in various stages of degeneration or necrosis with loss of cross-striation in most cases. There was quite considerable longitudinal striation, although a large number of the muscle cells were amorphous and eosinophil. There was no fibrosis or inflammatory cell reaction. The appearances in these sections suggested the ischaemia analogous to Volkmann's ischaemia, possibly at an early stage before replacement reaction had taken place."

Comment

(1) In this case I believe a secondary haemorrhage occurred when the foreign bodies were removed. (2) The increasing tension within the forearm deep to the flexor digitorum sublimis caused mechanical contracture of the fingers. (3) Relief of tension with splinting of the fingers in full extension permitted recovery to occur. (4) Without surgical treatment, replacement fibrosis of the necrosed muscle fibres would have "fixed" the existing contracture, and as cicatrization occurred this existing deformity would have become more pronounced.

I have to thank the Medical Superintendent of the hospital for permission to publish this case.

Medical Memoranda

Locked Twins

Though not quite as rare as was previously thought, a case of locked twins seems of sufficient interest to merit publication.

Case History

Mrs. A., a primipara aged 30, expected her first baby on Dec. 22, 1943. I saw her for the first time on Nov. 27, for obstructed labour. During the previous three weeks her pregnancy had been mildly toxaemic; there had been oedema of the legs and abdominal wall, a systolic blood pressure of 150 mm. Hg, but no albuminuria, headaches, or ocular symptoms. Her pelvic measurements were normal. Labour started naturally on Nov. 26, 36 hours before I saw her; six hours previously she had been found to be fully dilated, and for four hours she had been making strenuous efforts to deliver herself.

On examination she was found to be getting tired, though by no means exhausted. On palpating the abdomen one child was thought to be present lying in the left occipito-posterior position. Limbs were felt anteriorly, and one foetal heart only was heard—in the left flank. Palpation was made difficult by the oedema of the abdominal wall. An anaesthetic was then administered; vaginal examination after catheterization revealed that the membranes were intact: these were ruptured, and the hand introduced to confirm the diagnosis of persistent occipito-posterior position. The head, however, was found to be in an occipito-anterior position. To ascertain the cause of non-descent the hand was inserted further into the birth-canal, and it was then found that a second head was lying in the brim of the pelvis, wedged between the head and shoulders of the first child and completely preventing the birth of either. It was found possible to push up the head of the second child about an inch: forceps were then applied to the head of the first child, which was delivered with little difficulty. The second child was then delivered with forceps; it was also in an occipito-anterior position, and delivery was even easier than the first. Very little liquor amnii escaped. There were no lacerations, and a single placenta was expelled ten minutes later. Post-partum haemorrhage was normal in amount. The uniovular boy babies weighed 5 lb. 6 oz. and 5 lb. 2 oz. respectively.

The puerperium was normal, the mother feeding both babies at the breast; their weights on discharge from hospital were both 5 lb. 4 oz. The blood pressure returned to normal, and the oedema subsided during the first few days.

The frequency of locked twins is said to be 1 in 90,000 deliveries—i.e., once in about every 1,000 cases of twin birth. This statement was made by von Braun of Vienna, but in view of the number of cases recorded during the last ten years it seems

that the condition is not quite so uncommon. It is indeed surprising that locking does not occur more often. Oligohydramnios has been observed in many of the recorded cases, and seems the most likely cause: it was undoubtedly present in the case reported above, in view of the fact that, though there were two children weighing over 5 lb. each, the size of the abdomen did not suggest to anyone who saw the case that there might be a twin pregnancy.

I have to thank Dr. A. G. Winter, of Crowborough, for permission to publish this case.

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Latent Fractures of Vertebral Column after Aircraft Crashes

Three cases of fracture of the vertebral column after forced landings of aircraft were observed in a Polish Fighter Squadron within three weeks. Two of the accidents had occurred within a week of one another, and the third ten weeks previously. In each case there had been a latent period, varying from nine days to nine weeks, between the crash and the appearance of symptoms suggestive of fracture.

HISTORY OF THREE CASES

Case 1.—Flight lieutenant aged 30. Crash-landed on June 22, 1943. Sustained shock and severe cuts of the forehead. C.N.S. normal. Transferred to an E.M.S. hospital immediately after the crash and discharged as cured some weeks later. When on leave on Aug. 29 he felt a sudden pain in the back while digging in a friend's garden. His friend, who was a doctor, sent him for radiography, which revealed a crush fracture of the bodies of D.12 and L.1; this affected particularly the antero-superior border of each vertebra, the total vertebral border being only slightly reduced. This officer stated that he had had backache following the accident; but this had not been severe, and it had been vague in character. He was readmitted to hospital on Sept. 3 and discharged symptom-free on Sept. 29. No further symptoms have been complained of.

Case 2.—Flight sergeant aged 23. Forced landing on Sept. 2, 1943, with no visible injuries. Complained of pain in the right shoulder, which was *x*-rayed on the same day, no bony injury being apparent. There was no backache, and a physical examination revealed nothing abnormal. On Sept. 14 he complained of pain in the lower back, particularly on jumping from his aircraft to the ground. On examination there was some guarding of the lumbar muscles on the left, and he felt slight pain on bending the trunk to the right. Radiography on this date showed: fracture of right inferior articular process of L.5, obliquely upwards and outwards through the base; no displacement or deformity. He was admitted to hospital on Sept. 16 and was discharged symptom-free on Sept. 26. There has been no recurrence of symptoms.

Case 3.—Sergeant aged 27. Crash-landed on own aerodrome on Sept. 5, 1943. Had a cut of the head necessitating two sutures, otherwise no apparent disability. Detained in station sick quarters for 24 hours and discharged, after complete medical examination, without complaining of symptoms. On Sept. 14 he complained for the first time of pain in the lower dorsal vertebrae. Radiographs on the same day showed fracture of the inferior tip of the spinous process of D.7, with little displacement, and transverse fracture of the right inferior articular process of L.3, with no displacement. Detained in hospital until Sept. 20. Discharged symptom-free, and has not complained of any symptoms since.

COMMENT

All three fractures were anatomically different, although each was caused by a crash landing in the same type of single-engined fighter aircraft. These crash-landings were attended by varying degrees of damage to the aircraft, and by obvious injuries to the pilot varying from no visible injury in Case 2 to severe facial injuries and shock in Case 1. All three cases occurred among Polish personnel, although no specific significance can be attached to this.

A search in the literature has not revealed any similar occurrences. Rocher and Magnant (*Revue d'Orthopédie*, 1938, **25**, 694), report three cases of fracture of the vertebral column following crashes of gliders from low altitudes. They state that these fractures are common after glider crashes, being next in order of frequency to those of the legs and the head. Their cases also show three anatomically different fractures, although each of these lesions at once caused symptoms, making a fracture apparent or probable. All three patients likewise made full recovery, only one (where financial compensation was involved) showing any delay in the resolution of symptoms.

It is felt that a number of undetected fractures of the spine may possibly have occurred through crash-landings, and it is hoped that this note may draw attention to their possible occurrence.

Our thanks are due to Wing Cmdr. T. Montgomery for permission to publish this report.

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Reviews

FIELD SURGERY

Forward Surgery in Modern War. By Major-General W. H. Ogilvie, M.D., F.R.C.S., Hon. F.A.C.S., Hon. F.R.C.S.C., Army Medical Service. (Pp. 96; illustrated. 10s. 6d., plus 3d. postage; 5s. 6d. post free to doctors serving in H.M. Forces.) London: Butterworth and Co., Ltd.

"Field surgery," the author claims, "is the most important branch of military surgery because upon its quality all that follows will depend." With this there can be no argument. The need for a comprehensive work on the subject, based on hard experience in really modern warfare, has been felt for some time. That need has now been met, in the fifth year of war, by Major-General Ogilvie, who was consulting surgeon to the East African Force in 1941–2 and to the Middle East Forces from 1942 to February, 1944. The work, therefore, is an epitome of experience in East Africa, in Abyssinia, in the Western Desert, Libya, and Tunisia.

It is a slim volume, as any volume on field surgery should be, not merely for the pedestrian reason of easy carriage but because such a work should present principles rather than details. The latter, though important, will vary with every campaign, and field surgeons, not to mention administrators, must be adaptable. There is much also to be said for a work which is confined purely to field surgery, except for one chapter entitled "An Outline of Treatment at the Base." A good deal of confusion has been caused by articles, though not those written by men of genuine field experience, in which the advice given does not distinguish clearly or at all between field and base hospital surgery. Surgeons arriving fresh to the field after almost four years of war had much to learn. In the absence of casualties at home—even air-raid casualties are not really comparable—they presumably had taken their cues from published advice. The latter had been copious. Too much of it has been theoretical and "fancy." Yet if the brief papers published in this *Journal* in 1939 are re-read not much exception can be taken to the principles enunciated by men with long first-hand experience in the last war. Most of their observations have stood the test of time, even in a very different type of war; some of them, such as those of Col. J. M. Weddell on the possible drawbacks of air evacuation and on forward operating, have been prophetic. But military surgery does not stand still any more than any other art, and its further evolution is here clearly traced.

General Ogilvie's work gives a realistic presentation. There is no forgetfulness that the first object in war is to win battles, that fighting materials must have priority over medical stores, and that surgery must adapt itself to the campaign in hand. Thus, while his advice on the siting and organization of units, on the arrangement of operating theatres, and on the categorization of casualties is clear, it is also elastic. On technical points he not only gives his personal opinions but calls in the wealth of the Middle East Memoranda on Surgery on various subjects. These memoranda, under his own editorship and often authorship, and those of his predecessor in office, Major-Gen. D. C. Monro, not only have been a guide to Middle East surgeons but have been eagerly sought after and prized far and wide as the real substantial surgical advice in modern warfare. Thus we find also included here the advice tendered by specialists with field experience on resuscitation, burns, wound infection, neurosurgery, maxillo-facial surgery, and chest wounds.

What has the Middle East contributed to the art of field surgery that is likely to stand the test of other campaigns in different fields? In organization there have been the establishment of mobile surgical units and their exploitation to suit fluid warfare. Blood transfusion was, of course, making much headway in civilian practice, but its development and organization in the Middle East will surely serve as a model for the future. The employment of the sulphonamides has followed a similar pattern. In actual technical practices there are some changes unlikely soon to be discarded, even in the possible closer fighting of future campaigns. Among these there is the undoubted success of the Tobruk plaster for the transportation of the fractured femur patient and the like. There is the

conservative attitude in chest surgery. There are the practices of exteriorization of the injured colon and of almost routine gastric suction in abdominal wounds. Of the last two the former seems to have eliminated the condition of "colon septicaemia" which in the last war characterized the spread of infection to retroperitoneal spaces; the latter has greatly diminished the dangers of the almost inevitable post-operative ileus.

This book makes a timely appearance and cannot but add to the war effort in a large and very special way. Many surgeons new to field work will presumably soon be actively engaged. Without even a preliminary canter on the farthest fringe of a battlefield they will find much that is strange. What the written word can do to remedy the defect of inexperience has been done here. It is a lucid and attractively written work. Above all it bears the hallmarks of authentic knowledge and intimate experience.

YEARBOOK OF UROLOGY

The 1943 Year Book of Urology. By Oswald S. Lowsley, M.D., F.A.C.S. (Pp. 416; illustrated. $3.00.) Chicago: The Year Book Publishers.

Since research in urological problems has been disorganized by the war in a great many countries, few references are made in this edition to urological literature other than that published in the United States. But, the war notwithstanding, there is much to interest the reader in the 1943 yearbook. It contains descriptions of many new techniques in urological surgery. D. M. Davis's new operation for ureteral and uretero-pelvic stricture, which he calls "intubated" ureterotomy, is of great interest. It consists in splitting the strictured ureter longitudinally and wrapping the ribbon so formed round a large-sized catheter, which acts as a splint. Even though the ureter does not cover more than half of the diameter of its splint, a ureteral channel, normal in shape and size, will eventually be formed. To obtain this successful result it is essential that the catheter selected should be as large as will enter the uncut and presumably normal ureter and that it should be left in position for a minimum of three weeks. J. W. Lord, J. H. Eckel, and other researchers have been experimenting in a similar way with vitallium tubes in the ureters of dogs with good results. Another ingenious operation reported in the yearbook is the utilization of fascial strips obtained from the aponeurosis of the external oblique for reinforcing the female urethra in cases of stress incontinence. It sometimes happens that a surgeon is called upon to re-operate on a patient who has previously undergone an unsuccessful plication operation for this condition, and in such cases he often finds that the urethra has been so damaged that it is difficult to repair it. This difficulty can be overcome by the use of fascial strips according to the method suggested by A. H. Aldridge.

The 1943 *Year Book of Urology* is a very satisfactory publication, and supplies an easy method of revising recent advances in this important branch of surgery.

RADIOLOGY OF BONES AND JOINTS

The Radiology of Bones and Joints. By James F. Brailsford, M.D., F.R.C.P. Third edition. (Pp. 440; illustrated. 45s.) London: J. and A. Churchill. 1944.

The third edition of this standard work shows that the war has not restricted Dr. Brailsford's energies. The method of presentation remains unaltered: the first two-thirds describe abnormalities and diseases as they appear in individual bones, and the final third gives general descriptions of bone diseases. The author has kept a close eye on the international literature, and no observation of importance has escaped him. There are well over 1,000 detailed references, and it is obvious that each author's work has been critically analysed before being included in the text. Dr. Brailsford has also carefully followed up many obscure cases for a long period of years and thus has been able to bridge the gaps in previous editions. The result is a considerable increase in the size of the volume, which is now a veritable encyclopaedia of bone diseases.

It is not an easy book to read, and some of the descriptive terms must be incomprehensible to most doctors. Comparing the appearance of march fracture with a plumber's wiped joint is an obvious example. This, however, is the only flaw in an outstanding book which has no parallel in the English language, and should be kept for reference by every radiologist and hospital department. The illustrations are excellent, and special tribute must be paid to the author for personally compiling a first-class index.

Notes on Books

No. 23 of *Individual Psychology Medical Pamphlets* is published at 2s. 6d. by the C. W. Daniel Company, 40, Great Russell Street, W.C.1. This collection of papers suffers from the fact that its appearance has been very much delayed, most of the addresses having been delivered to the Medical Society of Individual Psychology early in 1940 before the war had well begun. After a foreword by Sir Walter Langdon-Brown a past chairman reviews the history of the society and its present chairman forecasts a future in which he hopes to see it amalgamate all other medico-psychological societies and found a common journal. A paper by Dr. Stekel describes how this one-time disciple of Freud has abandoned the sexual theory and admits that war neurosis may be the result of conflict between self-preservation and personal idealism; and it is interesting to note that his experience in the last war convinced him that the severe war neurotic could be cured only by excusing him future front-line service while retaining his self-respect by the fact that it was the doctor who took responsibility for this direction. Drs. Worsley and Barker discuss the approach to the patient, the first from the point of view of the modern common-sense eclectic psychotherapist, and the latter more from a Jungian standpoint. Next follows an excellent article by Dr. Dicks on analysis under hypnotics. He thinks it is useful in research into psychological mechanisms and in the reintegration of hysterics, but disappointing in the treatment of more severe cases. In a recent note he states that three years' further experience has not led to any modification of this view. Dr. Stungo adds a paper which, if rather more vague, comes to much the same conclusion, though he regards this form of therapy as a useful short cut in all forms of analysis.

Prof. JOHN ZAHORSKY assisted by Dr. T. S. ZAHORSKY has revised the popular *Synopsis of Pediatrics* (Henry Kimpton; 22s. 6d.) for a fourth edition. It is, indeed, a tightly compressed handbook in which the practical aspects of treatment are well stressed.

The Oxford University Press has published a shilling pamphlet for the Royal Institute of International Affairs entitled, *A Reading List for Relief Workers.* This bibliography, compiled in co-operation with some of the British voluntary societies most immediately concerned, is based on a selection of books that should prove useful to those who wish to take part in relief work in liberated territories. The publications are grouped under main headings.

Readers of previous editions of the *Year Book of Obstetrics and Gynecology* (Chicago: The Year Book Publishers; $3.00) will no doubt have ordered their copy of the 1943 publication. Those who are not familiar with the series would be well advised to start the *Year Book* habit right away. It is an excellent method of keeping in touch with recent advances, and the editorial comments by Dr. J. P. GREENHILL are a useful guide to the busy reader. The literature of the year does not always record progress, and when authors advocate practices which would be generally dangerous—as, for example, accouchement forcé or the use of pituitrin in labour—their views are summarized and the dangers are expressed in no uncertain manner. Among the advances of the last year the over-publicized subject of caudal anaesthesia receives considerable prominence, and many readers will agree with the editor that "despite its auspicious start, continuous caudal anaesthesia will not become part of our general obstetric armamentarium." Analgesia and anaesthesia in obstetrics, the Rh factor, hormone therapy in all its manifestations, human fertility, are but a few of the subjects on which useful information is to be found. This book is an excellent investment for the busy practitioner.

A 14th edition of *The Pharmaceutical Pocket Book* has been prepared by the Codex Revision Committee of the Pharmaceutical Society (London: Pharmaceutical Press; 10s., postage 4d.). This work is the lineal descendant of a pocket notebook for students of pharmacy first produced in 1906. In the course of its evolution it has come to serve a twofold purpose: to introduce to students of pharmacy the fundamental principles on which the work of their profession is based, and to give the practising pharmacist a handy reference book. Opportunity has been taken in the present revision to delete sections which are no longer considered useful to the student or pharmacist, and to modify and expand other parts. Among the many changes in the text and tables mentioned in the preface, additions have been made to the Dictionary of Synonyms and Trade Names (which occupies 76 pages) in order to bring the particulars up to date with respect to the *B.P.* Addenda, *B.P.C.* Supplements, and new proprietary preparations. The section on food and diet ends with a tabular list of vitamin contents of official preparations.

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THE MEANING OF "SHOCK"

On the eve of one of the greatest battles in history the medical profession of this country may well desire some clarification of the problem of shock. In our present issue an analysis is presented by Virgil Moon, and with many of his views workers in this field will be in complete agreement. Yet there remains a considerable diversity of experimental evidence or deduction therefrom, and it is worth while to attempt an analysis which will make the problem less confusing to the mind unfamiliar with the technical aspect. Many of the obstacles to clear thinking seem to arise from the use of the single term "shock." Moon points out that "shock" is a syndrome of multiple aetiology, and Lewis[1] has recently reminded us that "diagnosis is a system of more or less accurate guessing, in which the end-point achieved is a name. These names applied to disease come to assume the importance of specific entities, whereas they are for the most part no more than insecure and therefore temporary conceptions." Has the time come to discard this term? If "shock" is a disturbance of the circulation, then so is death; and it is as absurd to search for a single cause of the former as of the latter. If the term is retained it might be clearly understood that it represents only a convenient symbol for "low blood pressure associated with haemorrhage, burns, crushing injury, infection, cerebral injury, diabetic coma, coronary thrombosis, packed pulmonary embolism, etc." A telephone message that a casualty has been admitted in "shock" conveys very little; a message that the systolic blood pressure is 55 mm. Hg is a fact on which action can be taken. Authorities now agree that acute reduction of blood volume is of paramount importance in the low-blood-pressure states of casualties: first, because it is the commonest, and, secondly, it is the only cause which can be treated really successfully. They are far less agreed, and indeed are often silent, when it comes to explaining how a reduction in blood volume reduces the blood pressure. The usual explanation given is that diminished venous return causes diminished cardiac output. This explanation is meaningless in a closed elastic system in a steady state, for cardiac output and venous return are equal, and there is no means of telling which is the cart and which the horse.

Other methods of analysis, however, are becoming available. In the experimental animal the following important principle has been extensively used by Wiggers for study of blood-pressure changes: Mean blood pressure = cardiac output × total peripheral resistance. In man the methods developed by Cournand and his colleagues[2] allow serial estimation of cardiac output and thus the use of this formula. The analysis can be carried further, for though total peripheral resistance cannot be measured directly, blood flow through skin, muscle, brain, and possibly kidney can be estimated, so that an indication of the site of arteriolar vasoconstriction or vasodilatation may be obtained. Known factors affecting cardiac output are changes in venous pressure and heart rate, and substances that affect the heart directly. Thus the cause of a low cardiac output can be analysed by suitable physiological tests. For example, if the venous pressure is raised by a transfusion without any corresponding increase in cardiac output, then clearly the heart itself is at fault.

The data obtained by such methods are still scanty. Cournand and his colleagues[3] have published most interesting results in cases with low blood pressure after injury, and further important contributions are to be expected from this group of workers. It is already clear that mechanisms resulting in low blood pressure may be far more complex than the three suggested by Moon. Haemorrhage, for example, causes a steady fall in right auricular pressure and cardiac output, though blood pressure may be maintained, presumably by arteriolar vasoconstriction. The sudden fall of blood pressure which terminates a large venesection in man is apparently due to vasodilatation in muscle arterioles mediated by vasomotor nerves,[4] a reaction which may be identical with Moon's "primary or neurogenic shock." The steady fall in right auricular pressure on bleeding draws attention to reactions in the elastic venous system. If the pressure in the great veins is regarded as depending on the amount of blood in the venous system and the tone or tension of the veins, then it is clear that removal of blood can result in a fall of pressure only so long as venous tone does not increase. Slow changes in venous tone may, however, play an important part in the process of recovery, and may explain the surprisingly low circulating blood volume but normal systolic blood pressure found in a late phase after haemorrhage and in chronic anaemia. There is also the possibility that the sequence—low blood pressure, low cardiac output, low venous pressure—may result not only from a diminished amount of blood but from diminished venous tone.

The treatment of low-blood-pressure states at the present time is simple, for it consists solely of transfusion. Transfusion raises the venous pressure, and it is immaterial in practice whether the consequent rise of blood pressure results mainly from increased cardiac output or decreased arteriolar dilatation. If the blood pressure fails to rise after transfusion then there are three possibilities: first, the transfusion may be insufficient; this can be ruled out by simple clinical observation of the neck veins by the well-known methods described by Lewis, for if transfusion is continued until the veins of the neck show that the venous pressure is above normal, then no more can be expected from this method. Secondly, the heart may fail to increase its output on the raising of venous pressure; and, thirdly, the blood pressure may remain low from diminished peripheral resistance in spite of increasing the cardiac output. On failure of transfusion to raise the blood

[1] Lewis, *Lancet*, 1944, **1**, 619.
[2] *Proc. Soc. exp. Biol.*, N.Y., 1941, **46**, 462.
[3] *Surgery*, 1943, **13**, 964.
[4] Barcroft *et al.*, *Lancet*, 1944, **1**, 489.

pressure, attempts may be made to use other agents, such as constrictor substances, digitalis, and oxygen if arterial oxygen saturation is low. Such methods, however, are likely to prove disappointing. For this reason there may be some disagreement with Moon when he decries the need for new facts. In view of species differences, especially those connected with the erect posture, and the far greater ease of making the fundamental circulatory measurements, such new data may well come from controlled observations on man himself.

A NATIONAL PATHOLOGICAL SERVICE

We print elsewhere in this issue a memorandum, drawn up by a self-constituted group of leading pathologists, on the future of their specialty in a National Health Service. The White Paper, as they point out, has little to say on this aspect of medicine, and the arrangements by which it is to be enabled to play its proper part in an adequate medical service clearly call for consideration. These will not easily be made, for it is well known that present laboratory resources are quite inadequate; not only are they unprovided under the present system of National Health Insurance, but in many parts of the country distant from the larger cities laboratories staffed and equipped to undertake a wide range of pathological investigations do not yet exist. At the same time it is rather easier to visualize a national service for diagnostic pathology than for any other branch of medicine, because two examples of such a service on a limited scale have been in operation ever since the war began. The Emergency Public Health Laboratory Service, devised originally to reinforce existing safeguards against epidemics due to war conditions, has developed into a valuable instrument for organizing field investigations connected with epidemic disease and for encouraging prophylactic immunization, as well as providing greatly improved and extended diagnostic facilities. The system of reference laboratories, to which material for special and unusual investigations can be sent from any part of the country, is a good illustration of what can be achieved only by an organization on a national scale. The Sector system in London and the Home Counties is another example, more restricted geographically but far more inclusive in its scope. The Sector Pathologist, who is usually the pathologist in the principal London medical school in the Sector, is responsible for the staffing and administration of all laboratories in E.M.S. hospitals in his area. These sometimes include a Public Health Laboratory, and always laboratories part of the duties of which is to undertake any investigations required by private practitioners. Thus on occasion pathologists of academic standing who have eschewed private practice for years have been compelled to undertake it by virtue of holding a whole-time post under the Ministry of Health. Other E.M.S. pathologists, while perhaps not so unaccustomed to duties of this kind, have felt that they are thereby competing unfairly with their brethren in independent practice. In these areas, in fact, a national pathological service of a sort is already in being, and nothing but good can be done by stating plainly that some of those now compelled to work in it will strongly resist any attempt to retain them in such a service after the war. It is obviously wrong that the occupant of a university chair, whose main duties are teaching and research, should be saddled with administrative and other responsibilities extending far and wide beyond the walls of his own institution. Such an imposition must tend ultimately to the appointment of good organizers rather than brilliant academicians, to the great detriment of pathological science in this country.

The keynote of the memorandum on page 790 is co-ordination, and this, it is suggested, could be achieved by pathologists themselves, provided that the administrative structure permits it. There must be three main branches —teaching and research, clinical pathology, and public health bacteriology—requiring separate staffs. The two latter may well be fused in small centres serving a population of 100,000; in large areas they should be distinct, but may with advantage be housed in the same building. The university department which is apparently visualized as the core and centre of an area in a national scheme should retain its academic freedom, and the professors in charge "should take no part in the professional administration of the hospital laboratories in the regions, and while they should be closely associated with pathologists engaged in diagnostic and prophylactic work they should not themselves be responsible for it." These departments should also be "the training centres of sufficient pathologists to supply the needs of the service." The free interchange of professional and technical staff, particularly in hospital laboratories, is thought desirable "in order to encourage efficiency, to prevent isolation, and provide for study leave and sickness." For the wide administrative responsibilities entailed in directing such a service a special type of officer is contemplated, who should preferably hold also a part-time university appointment; this is "a post requiring knowledge, experience, and tact, and should not be held by anyone too junior to command respect or too senior to have lost touch with bench work." A democratic note is struck in the suggestion that a representative committee of regional pathologists should have a voice in his appointment. A committee within each area of the Joint Authority, as forecast in the White Paper, is also suggested, which shall be representative of all the laboratories in the area and advise the authority on pathological matters. Another important recommendation, unconnected with the main theme of the memorandum, concerns the conduct of coroners' necropsies. It is suggested that there should be a small number of laboratories specializing in medico-legal work in its criminal aspects, working in close touch with the Home Office and attached to a university. For necropsies where crime is not in question the services of a senior hospital pathologist should be secured. We agree that the sooner the general practitioner is relieved of this responsibility the better; it is not only irksome to him, but, except in the most straightforward cases, not seldom quite beyond his capacity.

These and other recommendations in this memorandum call for very careful consideration. They furnish by no means a complete scheme, but they indicate the lines on which a national scheme might work without disrupting

this branch of the profession, and without placing new burdens on some of its most valuable members. How the efforts of the three authorities concerned—university, Ministry, and local or regional authority—are actually to be co-ordinated except by the efforts of pathologists themselves the memorandum does not attempt to suggest. What is quite clear is that almost everything remains to be done before pathology can take its place in a national medical service. The more undesirable features of present arrangements have been accepted by those concerned only as a war necessity, and originally with the idea that peace would see a complete restoration of the *status quo ante*. If they are to be perpetuated and extended this must mean rebuilding from the very foundations.

THE STOMACH IN PERNICIOUS ANAEMIA

When reviewing recent work on the stomach in pernicious anaemia in 1940[1] we expressed the hope that we might be on the verge of far-reaching discoveries in alimentary physiology. This hope has unfortunately been disappointed. The suggestion[2 3] that the anti-anaemic factor might be secreted by the argentaffine cells of the alimentary tract was not supported by Magnus,[4] who found that these cells were often more numerous in pernicious anaemia than in health. Moreover, the pterins, which are the active principles of these cells, have not yet been clearly shown to have haemopoietic activity or to be related to the haematinic factor in liver extracts. Two recent papers do little more than confirm the view that the characteristic alimentary lesion in pernicious anaemia is destruction or atrophy of the secretory epithelium of the fundus and body of the stomach. Cox,[5] like previous authors, found that an outstanding characteristic was the contrast between the notable change in the mucosa of the fundus and body and the relative freedom from abnormalities in the pyloric zone. None of the findings varied consistently with the duration of the disease or the treatment, and they therefore do not support a suggestion by Wallgren[6] that inflammatory changes in the pylorus may have been removed by treatment with liver.

Wallgren found inflammatory exudation and degeneration of the glands in the pylorus in all his patients who had died before the introduction of liver therapy, though Brunner's glands in the duodenum were usually intact. This may be a disagreement about terms rather than findings, for there seems to be a fairly wide physiological range in the amount of infiltration of the interstitium and the degree of branching of the epithelium in the antrum.[7] Moderate degrees of simple atrophy and atrophic gastritis are so common, particularly over the age of 50, that they may almost be regarded as normal age changes. Gastroscopy is not of much value in determining the precise nature of the lesion in pernicious anaemia, for, although atrophic gastritis is characterized by a pale thin mucosa with a network of easily visible blood vessels, the gastroscopist fails in many cases to recognize proved atrophy of the antral mucosa.[8] Nevertheless, there is no doubt, even from Wallgren's own material, that the morbid anatomical lesion in pernicious anaemia may be entirely limited to the body of the stomach. Biochemical studies of the disorder are still rather contradictory. Examination of the livers of patients dying from cancer of different parts of the stomach suggested that they had least haematinic activity when the antrum was destroyed.[9] On the other hand, the results with desiccated stomach from different parts of the human stomach show that the haematinic activity resides principally in the body, and its situation therefore coincides with that of the anatomical injury.[10] In the pig the findings appear to be the exact reverse of those in man. The most active anti-anaemic preparations are those obtained from the antrum of the stomach.[11 12] On the other hand, while it is not possible to produce pernicious anaemia in the pig, selective resection of the body of the stomach causes complete disappearance of the P.A. factor in its liver. If the cardia is left behind, parenteral treatment with nicotinic acid will restore the P.A. factor in the liver. This has led to the tentative hypothesis that in the pig the fundus brings about the liberation of nicotinic acid from the food and its absorption, and that this substance then, by way of the blood stream, causes the cardia, or possibly the cardia and fundus, to form the active liver principle.[13]

OESOPHAGEAL SPEECH

The use of radiotherapy for cancer of the larynx has shown consistent success only in early cases which would be suitable for laryngofissure. In the more advanced cases radiotherapy has proved an uncertain and often a disastrous remedy, so that there is a large residue of cases in which laryngectomy is the only means of securing any reliable result, the effect of irradiation in such cases being a gamble, with the odds heavily weighted against the patient. The operation itself has now a low mortality, with a convalescence that is not distressing, though many still think of the effects as produced under the conditions which prevailed at the end of the last century. It is nevertheless a mutilating operation which must be justified, because it entails the suppression of the normal voice. At first this loss was overcome by a prosthetic apparatus which supplied a sound to replace the lost phonation, and the sound conducted to the mouth by a tube was turned into articulate language by the lips, mouth, and tongue. Many patients still use this artificial larynx, which with practice enables them to talk clearly, audibly, and continuously. A telephone conversation is quite easy. It has long been known, however, that a few patients were able to produce a real voice with the pharynx and oesophagus, something much more than the indistinct vowel-less whisper yielded by attempts at articulation without any phonation.[14] These patients were regarded as lucky freaks of Nature, but a study of the manner in which these persons produce their voice has enabled a large proportion of those who do not acquire the trick spontaneously to be trained in voice production and to acquire a good voice without any prosthetic apparatus. An elaborate study by means of *x* rays was made by Prof. Burger of a man who was positively able to shout, although rather hoarsely.[15] It was seen in this man that air imprisoned in the stomach and lower oesophagus could be forced out at will by the diaphragm, and this belching produced a sound which could be transformed into almost normal speech. Both the air bubble in the stomach and the air in the lower oesophagus were controlled by a pseudo-glottis formed at the upper end of the

1 *British Medical Journal*, 1940, **1**, 219.
2 Jacobson, W., *J. Path. Bact.*, 1939, **49**, 1.
3 Wilkinson, J. F., *British Medical Journal*, 1940, **1**, 414.
4 Ibid., 1940, **1**, 415.
5 *Amer. J. Pathol.*, 1943, **19**, 491.
6 *Acta med. scand.*, 1943, **115**, 365.
7 Schindler, R., and Ortmayer, M., *Amer. J. digest. Dis.*, 1942, **9**, 411.
8 Benedict, E. B., and Mallory, T. B., *Surg. Gynec. Obstet.*, 1943, **76** 129.
9 Schenken, Stasney, and Hall, *Amer. J. med. Sci.*, 1940, **200**, 11.
10 Fox, H. J., and Castle, W. B., ibid., 1942, **203**, 18.
11 Henning, N., and Burgsch, H., *Dtsch. med. Wschr.*, 1931, **57**, 757.
12 Meulengracht, E., *Z. klin. Med.*, 1936, **130**, 468.
13 Petri, S., *et al.*, *Acta med. scand.*, 1944, **116**, 273.
14 Thomson, StClair, and Colledge, L. *Cancer of the Larynx*, Kegan Paul, London, 1930, p. 188.
15 Burger, H., and Kaiser, L., *Acta Otolaryngol.*, 1925, **8**, 90.

oesophagus, presumably from the edges of the two inferior constrictors. The pseudo-glottis is situated at the level previously occupied by the cricoid cartilage, and can sometimes be seen with a laryngoscope as two well-defined bands which can be made to vibrate at will. In other cases these bands appear less defined than would be expected from the quality of the voice.

From a surgical point of view the opening made into the pharynx after turning forward the larynx is better closed vertically, with two short prongs to the fork at the upper end, than by a horizontal closure.[16] Gatewood suggests that this vertical closure may also encourage a better functional adaptation of the inferior constrictors to the formation of the pseudo-glottis.[17] McCall has recommended that the necessary voice training should begin several days before the operation, while the larynx is still in place, and has cited some figures to show that the voice is thus regained more quickly and more easily. However, Woods pointed out many years ago that the physical difficulty of the act of belching to order is much less after the larynx has been removed, because the cricoid cartilage is no longer in close contact with the posterior wall of the gullet, and considerable effort is needed to overcome the force with which the cricopharyngeal part of the inferior constrictor normally holds the larynx back.[18] It is doubtful, therefore, if anything is gained by preliminary training, but the services of an expert in voice production are invaluable after healing is complete. The details of this lie within his province, but he can be greatly assisted in his task by not allowing the patient to whisper without attempting to produce a voice. If he speaks only when he attempts to produce a voice he will much sooner acquire the habit of speaking in this unnatural way. He no longer depends for voice production upon air from the lungs but from the oesophagus; consequently the close association between speech and breathing has to be broken, and it is much harder to break this instinctive association if the patient reverts to it by whispering between the acts of pharyngeal phonation.

REHABILITATION FILMED

"Accident Service," the second medical film produced by the British Council, was recently shown to a medical audience in London at the Curzon Cinema. The film was made at the Mansfield General Hospital and Berry Hill Hall Rehabilitation Centre, which together provide a highly effective machinery for dealing with accidents in the neighbouring coalfields. In the opening scenes the problem of accidents in highly industrialized Britain is briefly stated with the aid of diagrams, and it is shown that coal-mining is the most hazardous of industries. A miner who has been injured by a fall of rock is brought to the surface by his comrades, who are expert first-aiders. He is taken by ambulance to the hospital, where a fractured spine is diagnosed. A plaster cast is applied, and after a week in bed the miner is able to start walking in the ward. Even during this brief period in bed a planned scheme of exercise is instituted, and there is a progressive increase in the range of active movements, including occupational and recreational therapy, until, with the removal of the plaster, the miner is ready for transfer to the rehabilitation centre for the final stages of his treatment. Miners suffering from other injuries are also shown in different stages. The differences in the mechanism and treatment of a fracture and a fracture-dislocation of the spine are vividly depicted by animated diagrams. Another sequence shows the technique of wound excision with later skin-grafting in a case of compound fracture of the tibia and fibula reduced under direct vision.

These diagrammatic and operating sequences leave little room for doubt about the part films could play in surgical teaching. "Accident Service" provides a powerful argument for the use of the film in medical education, particularly in regard to those subjects which cannot be learnt adequately from the printed or spoken word and are outside the immediate hospital experience of the student. Although the word "rehabilitation" has now won such general acceptance that it is in danger of becoming a political slogan, there are few who could give a convincing definition of it. This film, which shows in some detail the actual working of one of the pioneer rehabilitation centres, succeeds in conveying what it is so difficult to define in words. Rehabilitation is not a method of treatment but a regimen. The fundamental idea behind rehabilitation is at least as old as the Hippocratic writings. Throughout the history of Occidental Medicine there has been a recurrent struggle between the natural followers of Hippocratic Cos and those of Cnidus. Cnidians, ancient and modern, treat the *organ* or assail the *disease*. "For Coans, then and now, *disease* is a dissociation of functional unity of the whole organism: their therapeusis is directed towards re-establishment of functional integrity."[1]

Another reflection prompted by this film is that rehabilitation is limited by the quality of the primary treatment. In other words, good surgery is still indispensable. Sir Alfred Webb-Johnson, in an introductory speech, pointed out that whatever value might justly be placed upon the personal doctor–patient relationship, the film demonstrated that the injured miner must have an accident organization behind him. This impression will have been shared by most of the audience, but they will have been equally impressed by the special skill, experience, and sympathetic understanding of the mind and life of the miner that have been necessary for the creation of the rehabilitation centre depicted. Sir Alfred paid a generous tribute to Mr. E. A. Nicoll, the surgical director of the centre, and said that his work was a credit to British surgery. The additional point may be made that rehabilitation centres such as Mr. Nicoll has evolved over a period of years cannot be created overnight by official circulars, although his work and that of other pioneers in the same field have provided a splendid foundation upon which others may build.

A MEDICAL NOBEL INSTITUTE IN SWEDEN

The Royal Caroline Institute (Kungl. Karolinska Medico-Kirurgiska Institutet), which is the medical school of Stockholm—the university (Stockholms Högskola) having no medical faculty—and is in charge of the medical Nobel Fund, has decided to build a Medical Nobel Institute for research in the three theoretical disciplines of anatomy, biochemistry, and physiology. The Institute will consist of three departments in one building to be erected in the grounds of the new medical centre at Norrbacka in the north-west region of the city. The biochemical Nobel Institute was founded in 1937 and is directed by Prof. Hugo Theorell. The physiological department will be a neurophysiological research laboratory privately endowed in 1940 for Prof. Ragnar Granit, who will also be in charge of the new institute. The anatomical department will be connected to a new chair in cytological research to be established for Dr. Torbjörn Caspersson.

[16] Colledge, L., in *Maingot's Postgraduate Surgery*, Medical Publications, Ltd., London, 1937, **3**, 4893.
[17] *Southern med. J.*, 1943, **36**, 453.
[18] *Arch. Otolaryngol.*, 1943, **38**, 10.
[19] *Surg. Gynec. Obstet.*, 1922, **34**, 303.

[1] Crookshank, F. G., in Cumston, C. G., *An Introduction to the History of Medicine*, London, 1926.

PATHOLOGY AND THE NATIONAL HEALTH SERVICE

A group of pathologists under the chairmanship of Prof. S. P. Bedson, F.R.S., have drawn up the statement printed below on some aspects of pathology in relation to the proposals in the White Paper.

1. Pathology, except for a brief reference to public health bacteriology and to "laboratory service," is not mentioned in the White Paper, and we feel it is time that constructive proposals regarding the position of pathologists and the part which pathology ought to play in the new medical service should be made by practising pathologists. We have no mandate to speak for our fellows or for any administrative authority, but we have reason to believe that our views accord with those of the majority of our colleagues.

2. We believe that the closest co-ordination between the two main branches of pathology—academic and applied—is essential to a satisfactory pathological service. Nothing should be allowed to split the component parts into a number of watertight compartments.

3. Academic pathology, mainly concerned with teaching and research, will inevitably be centred in the medical departments of the universities, and will, we suppose, be fully considered in the report of the Interdepartmental Committee. It would, however, be undesirable were research and teaching to be confined to university departments and research institutions. The efficiency of laboratories chiefly concerned with routine work can be ensured only if their staffs have opportunity for independent investigations, and for benefiting by the stimulus that teaching offers. For this reason the staffing of hospital laboratories must be adequate enough to afford the pathologists time to make use of these opportunities as they arise.

4. It may prove expedient, at any rate for the time being, to place what is called public health bacteriology under a separate administration. Whilst we should regret this, we believe that the detrimental effect on pathology as a whole which such a schism would entail could be mitigated to a considerable extent if adequate co-ordination with the rest of pathology could still be maintained.

5. Clinical pathology is an important part of a general hospital service, and as such must be closely bound up with the general scheme of hospital management. But a brief consideration of the technical practices of clinical pathology and public health bacteriology shows that they are so closely related as to make division unprofitable.

6. The co-operation of the two main branches of pathology may be seriously handicapped by being administered by three different organizations—university, Ministry, and local or regional authority. The desired co-ordination of the parts into one whole, however, can be achieved primarily by the pathologists themselves, provided that the scheme of administration is such as to permit of this.

7. Within the areas of the Joint Authority as envisaged in the White Paper there should be pathological committees capable of advising the Joint Authority on all pathological matters. These committees should be representative of all the laboratories in the area, and constituted in a similar manner to those which are already working satisfactorily in some of the defence regions.

8. The university departments of pathology should retain their primary functions of teaching and research, and act as the training centres of sufficient pathologists to supply the needs of the service. The professors should act as the friends and advisers of all pathologists in their regions, and, in some cases, as national advisers on special subjects and research problems. They would take no part in the professional administration of the hospital laboratories in the regions, and while they should be closely associated with pathologists engaged in diagnostic and prophylactic work they should not themselves be responsible for it.

9. The pathologist in charge of the clinical laboratory at the university hospital should, together with appropriate members of his staff, hold university appointments and take some part in the teaching of pathology: he would not ordinarily be responsible for the clinical pathology from outside hospitals or general practitioners. Such matters as interchange of staff between the university departments and the regional laboratories, the recognition by the university of senior pathologists in the region, and the extension of consultant and laboratory facilities to them would be a matter for detailed arrangement with each university. Several of the provincial universities have already made such arrangements.

10. The public health bacteriological service can only function as a separate administration at its main centres, and even there it must be closely associated with the universities and the larger hospital laboratories, if only to avoid uneconomic duplications of laboratories and staffs. At the periphery there must be complete fusion. A town serving an area containing less than 100,000 inhabitants cannot support more than one laboratory with reasonable economy and efficiency. In larger towns both the hospital and public health laboratories should be housed under the same roof even though under separate administration. There should also be laboratories solely devoted to public health and sufficient in number to provide for field work and epidemiological control throughout the country. In our opinion the population covered by such a laboratory would be from 3 to 4 millions. Provided that the hospital pathologist keeps the public health centre fully informed of all events appertaining to epidemic disease there is no need for any greater divorce of public health bacteriology from hospital pathology.

11. Similarly there should be a small number of laboratories specializing in medico-legal work in its criminal aspects, working in close touch with the Home Office and attached to a university. The majority of coroners' cases are not concerned with criminal action, and should be referred to senior hospital pathologists, but not to general practitioners.

12. The clinical laboratories must be situated in hospitals, under the disciplinary and financial, but not professional, administration of the owning authorities. The remarkable diversity of arrangements under which a professional man may now act as pathologist to a hospital should be brought to an end. The pathologist in charge should be of consultant rank, appointed on precisely the same terms and at the same rate of pay as his clinical colleagues, and serving with them on the medical council of the hospital.

13. Laboratory facilities must be freely at the disposal of the general practitioners in the area, but there should be no isolated small laboratories staffed only by technicians or a solitary pathologist: small laboratories should be under the surveillance of a large hospital laboratory in the vicinity which would provide the staffing. This would ensure a continuous linkage between the rural practitioner desiring pathologist assistance and the research laboratory in the university, without imposing an undue burden of technical or administrative responsibility on any single link.

14. Professional co-ordination of all laboratory services within a university region is necessary for smooth working, and experience in the Emergency Medical Service has shown that this cannot in normal times be the function of a professor or of the full-time director of a laboratory. The person responsible has to act as an intermediary between the pathologist and the administration, making it his business to see that every pathologist has all possible facilities and acting as adviser to the administration. It is a post requiring knowledge, experience, and tact, and should not be held by anyone too junior to command respect or too senior to have lost touch with bench work. Such an individual might well hold a part-time post in a university department, and a representative committee of the regional pathologists should properly have a voice in his or her selection. We regard a post of this sort as essential to a proper co-ordination of the service.

15. The hospital laboratories at present in existence have provided only a skeleton service for the country, and a larger number will be needed to provide for the needs of mental hospitals, fever hospitals, and sanatoria, as well as general hospitals.

16. It is in hospital pathology in particular that a free interchange of professional and technical staff is essential, both within a large region and outside it, in order to encourage

efficiency, to prevent isolation, and provide for study leave and sickness. Local hospital administration will present financial difficulties to this interchange, which are likely to require central adjustment of a kind which has, on the whole, proved sufficiently elastic to solve similar difficulties in the Emergency Medical Service.

17. While the professional freedom of individual pathologists, as of other practitioners, must be maintained, we firmly believe that some factual, and not merely theoretical, central control of the whole service of pathology will have to be exercised if it is to be conceived as one complete section of medical service to the community. If this conception fails to be a matter of Government concern, medicine will, in this important respect, be moving backwards instead of forwards.

S. P. BEDSON. P. N. PANTON
T. B. DAVIE. A. H. T. ROBB-SMITH.
S. C. DYKE. A. F. S. SLADDEN.
H. B. MAITLAND. JANET M. VAUGHAN.
A. A. MILES.

Reports of Societies

DISINFECTION OF AIR

At a meeting of the Section of Epidemiology and State Medicine of the Royal Society of Medicine held at the National Institute of Medical Research, Hampstead, under the presidency of Sir WELDON DALRYMPLE-CHAMPNEYS, a demonstration was given by Dr. R. B. Bourdillon and his colleagues, Dr. O. M. Lidwell, Mr. J. E. Lovelock, and Mr. W. F. Raymond, on the study of air-borne bacteria and methods of air disinfection.

Dr. BOURDILLON explained that at the beginning of the war certain workers under Sir Patrick Laidlaw were engaged at the Institute on this study and showed that a number of methods could be used with good effect against the influenza virus. The work was afterwards taken over by Dr. C. H. Andrewes, who later turned his attention to typhus, and only a section of the former team now remained; but he hoped some useful work had been done. Four factors might be said to dominate air disinfection as seen in field trials and in actual practice. The first was the fact that while people were inclined to take an interest in the installation of any new system of air purification the interest was liable to give place to boredom and the system to fall into disuse. The second was that clinical trials were difficult, except in isolated communities, on account of the many sources of respiratory infection, so that there might be little to show for one's labours. The third was that the particles in which they were most interested, as a rule, were virus particles, and the methods available for their detection were not suitable as yet when the particles were scattered in very dilute concentrations in the air of rooms. Finally, air disinfectants were rather "messy" and troublesome, and concentration had to be carefully controlled. The whole subject must be regarded as still in the experimental stage, and it was well to start on a small scale and in places where there was a special reason for such a system. Dr. Bourdillon placed the methods available in the following order of preference:

(1) Good ventilation and avoidance of overcrowding.
(2) Daylight, whenever conditions permitted. Recent work had shown the value of daylight, even after passing through window glass, in keeping down bacteria.[1]
(3) Dust control. There was accumulating evidence that dust-borne infection played a very important part.
(4) Ultra-violet irradiation, which was to be recommended on account of its cleanliness.
(5) Various chemical methods suitable in special cases.

Chemical Methods

The chemical methods included the use of hypochlorous acid gas, of propylene glycol (which had been widely studied in America and to some extent in this country), of triethylene glycol (which was effective only within a narrow range of humidity, and therefore could not be recommended for general use), of resorcinol (which was effective against freshly sprayed moist bacteria, but disappeared from the air rather rapidly, so that in order to maintain a steady concentration it was necessary to vaporize considerable quantities), and of lactic acid, which he thought might have a future on the ground of its cheapness and the fact that it was effective over a wide range of humidity. The efficiency of all these agents for killing moist bacteria and virus particles freshly sprayed into the air from cultures or from the respiratory tract depended greatly on the relative humidity of the air. Ultra-violet light showed reduced efficiency at relative humidities of over 70%, but increased in efficiency down to at least 40% relative humidity. Propylene glycol required a humidity between 35 and 75% to be of much practical use. Resorcinol and hypochlorous acid became inefficient below 45% relative humidity, but worked well against freshly sprayed moist organisms at 60 to 70% relative humidity (and probably higher values) at normal room temperatures. Lactic acid was effective against organisms freshly sprayed in saliva at humidities from 30 to 80% or more. In concentrations suitable for daily life many of these agents were relatively inefficient against spores and the dried saprophytic organisms found in dust. Hence if Petri plates were exposed in crowded rooms the number of viable organisms collected might be little affected by these agents unless excessive concentrations were used. As most of the organisms thus found were not pathogenic, this apparent failure to kill might be misleading. Tests appeared to show that lactic acid was better than the other agents in this respect. If air disinfection was needed suddenly on a large scale to meet an epidemic or other emergency, the most practicable method was the evolution of hypochlorous acid gas by boiling a solution of bleaching powder in water with an acid phosphate. (A demonstration of this was given.) If a relatively small number of dwellings were to be disinfected, either propylene glycol or ultra-violet light was probably the safest agent, but lactic acid might be worth trial. The reduction of dust by the use of mineral oil on floors of wood or linoleum was simple and economical and seemed to be one of the most important measures now practicable on a large scale.

Air disinfection was easy in the laboratory but troublesome and expensive when used on a large scale, and therefore inferior to good ventilation when this was practicable. The chief problems were the production of adequate quantities of the bactericidal agent, its distribution in tolerably uniform concentration, the securing of suitable humidity in the air, and the high resistance of dry dust-borne organisms.

Discussion

In answer to a question whether there was any prospect of evolving a smaller or more compact apparatus for vaporizing lactic acid Dr. BOURDILLON said he hoped that in the future substances would be available which were easier to vaporize than lactic acid; but lactic acid could be used quite cheaply with a hand spray, and it was this he recommended for offices or small buildings. Sir LEONARD ROGERS asked whether anything could be done with the vaccinia virus. Dr. C. H. ANDREWES said it was possible to kill vaccinia in the air with small sprays. Dr. MERVYN GORDON said that vaccinia was very susceptible to permanganate of potash. This substance inhibited vaccinia and smallpox virus within half an hour. The late Andrew Balfour, when faced with a big epidemic of smallpox in the Sudan, controlled the scarring completely by saturating lint with permanganate of potash and applying it to the skin. His idea had been to kill the secondary infection, but what he really did was to inactivate the primary. Permanganate seemed to work in the same way with other viruses—that of poliomyelitis, for example.

In reply to other questions Dr. ANDREWES said that the envelope surrounding dried particles was a very great mystery. Propylene glycol seemed to be singularly inefficient when it came to organisms with dust, but it looked as if lactic acid might be more effective. Questions were asked on the ventilation of railway carriages. Dr. BOURDILLON considered that it would be better to ventilate than to use chemicals for disinfection, since the opportunities for ventilation were unlimited. Dr. J. A. H. BRINCKER asked whether ozone was discredited. Dr. BOURDILLON replied that very careful tests had recently been carried out by Dr. Elford at the Institute on that very point. Ozone was useless as an air disinfectant. In order to get an efficient killing of air-borne bacteria it would be neces-

[1] *British Medical Journal*, Feb. 19, 1944, p. 245.

sary to have a concentration of ozone which was likely to do more harm than good. He was not speaking of water-borne infection.

One of the demonstrations carried out in the course of the afternoon concerned the effect of lactic acid on air-borne bacteria in a crowded mess-room. With no method of disinfection the number of bacteria-carrying particles per cubic foot rose, on the average of a four-day experiment, during the midday period when the room became crowded, from some 15 to nearly 40, and continued at that level for half an hour, gradually subsiding to the original figure as the room emptied. When lactic acid was used, the number of particles per cubic foot, on the average of a three-day experiment, never rose higher than about 18, and dropped to 12 even while the room continued to be crowded, subsequently falling to about 5. The reduction in the number of bacteria-carrying particles due to the presence of lactic acid was estimated at 69%. The conditions during this experiment were: relative humidity, 49–75%, temperature, 16–21° C.; lactic acid concentration, 0.08–0.11 mg./c.ft. of air.

DIETARY SURVEYS

A conference of the Nutrition Society was held on May 20 at the London School of Hygiene and Tropical Medicine, with Dr. JOHN HAMMOND, F.R.S., in the chair, for a second discussion on budgetary and dietary surveys of families and individuals. [The earlier discussion was reported in these columns on March 18, p. 401.]

Difficulties of Assessing Intake

Miss E. M. WIDDOWSON and Dr. R. A. MCCANCE, of Cambridge, explained their methods and difficulties in conducting individual dietary surveys. They insisted that in the collection of data all food items should be weighed before being eaten, and doubted the accuracy of the "budgetary" and "questionary" methods. Individuals should be studied rather than families in view of the inaccuracy involved in converting data for women and children into "man values." Food consumption varied widely even in individuals of the same age and sex. Thus the individual daily food intakes of a group of 14-year-old girls lay between 1,708 and 3,736 calories. A further complication in the use of "man values" arose from the irregular variation of the intake of various nutrients with age. It was true that the protein intake increased fairly smoothly from 40 g. for boys and girls at 1 year to 80 g. for girls and 100 g. for boys at 16 years. The calcium intake, on the other hand, remained steady, because the very young, although eating less, drink relatively more milk. The vitamin C consumption might actually fall, because orange juice was given most freely in infancy. Equally formidable difficulties arose in the application of food tables to convert the data collected in terms of foodstuffs into individual nutrients. Investigators had sometimes made the grave error of taking data for uncooked foodstuffs, particularly vegetables, as applying to the cooked article. Calcium intakes could not be calculated accurately from food tables in the London area because of the high calcium content of the water supply. The iron content of foods might be greatly affected by the nature of the cooking utensils. Thus apples cut with a stainless knife and cooked in a glass vessel were found to contain 0.31 mg. of iron per 100 g., while apples cut with an ordinary knife and cooked in an iron vessel contained 6.0 mg. per 100 g.

Prof. V. H. MOTTRAM suggested that in view of the serious criticisms raised by the previous speakers against both the indirect calculation of calorie requirements and the use of "man values," it might be necessary to revert to the calorimeter. Although this instrument was expensive and laborious in use, it might be better to face these objections rather than take quick methods which gave wrong results. Dr. F. C. HAPPOLD reported a survey among students in Leeds, in which mean calorie intakes of 2,609 and 2,640 were found on different occasions. Mr. A. L. BACHARACH emphasized the danger of confusing consumption with requirement. The mere fact that children were given a certain allowance of milk did not imply that they needed it.

"Science" of Cooking Vegetables

Dr. GERTRUDE WAGNER, of the Wartime Social Survey, described her extensive investigation into the methods used by housewives throughout the country in cooking vegetables. The effect of the methods most commonly used on the destruction of vitamin C was studied at the Dunn Nutrition Laboratory, Cambridge. To avoid loss of vitamin it was important not to have too much cooking water, not to cook too long, and to eat the vegetables soon after they were cooked. Mrs. B. CALLOW pointed out that whereas in America the abundance of citrous fruits left only 50% of the vitamin C requirement to be derived from vegetables, in this country vegetables must provide 80% of our requirement. There was room for improvement in cooking vegetables.

Dr. G. N. JENKINS, Dr. L. W. MAPSON, and Miss M. OLLIVER described the chemical methods they had worked out for the estimation of nutrients in meals collected at canteens in the Cambridge area. The importance of adequate sampling was emphasized, and as a demonstration a complete meal was homogenized in a Waring blender in a matter of seconds. The agreement between values found by analysis and calculated from tables was generally fairly good. Dr. C. P. STEWART, of Edinburgh, considered that chemical analysis was not usually necessary, calculations based on tables being accurate enough for most purposes. He gave, however, a striking example of the loss of vitamin C in the preparation of vegetables for hospital patients. The raw vegetables contained 14–41 mg. per 100 g., when freshly cooked 10–38 mg., and when finally reaching the wards only 0.7–3.6 mg.

Success of School Meals

Miss E. M. LANGLEY outlined the activities of the Board of Education in supplying meals for school-children. She recounted how the first steps had been due to charity. Thus in 1862 Victor Hugo supplied forty poor children in Guernsey with meat and wine. Official action might be dated from the Provision of Meals Act in 1906. The dinners then provided were a monotonous repetition of soup or hash, bread, and rice pudding, and cost about 1d. The meals now served in elementary schools were designed to supply about 1,000 of the child's daily requirement of 2,500 calories, and on an average contained 20–25 g. of protein, 30 g. of fat, 2,450 i.u. of vitamin A, 230 i.u. of vitamin B_1, 54 mg. of vitamin C, 5.7 mg. of iron, and 425 mg. of calcium. One ounce of dried milk was allowed for every meal, which supplied about half the protein requirement. Sliced raw vegetables were given with "conveyed" meals to make good the deficiency in vitamin C. The meals usually cost about 5d., and were varied and attractive. The "Oslo dinner" of milk, brown bread, cheese, salad, and an orange was popular with children, but took a long time to eat. About 1,500,000 children were now fed at school, as compared with 250,000 before the war.

Dr. M. PYKE, of the Ministry of Food, spoke of the food provided by industrial canteens. The total daily intakes at work and at home for various classes of operative were estimated by questioning individuals as to their consumption over the last 24 hours, portions of food of known size being displayed for purposes of comparison when necessary. It was found that as many as six meals a day might be taken when snacks of 200 calories or over were included. For Lancashire women cotton operatives the daily intake was 2,460 calories and for male "mule" spinners 4,030 calories. A typical midday canteen meal supplied 850 calories if all was taken, but workers usually took only 630 calories, which was, however, more than the 510 calories they got at home. The proportion of the daily requirement eaten in the canteen depended on the times of shift. Men "knocking off" at 3 p.m. ate only 35% of their daily intake in the canteen, as compared with 55% for men who finished work at 5.30 p.m. More food was eaten in two sandwich meals taken during the shift than in one hot conventional meal with meat and potatoes. Coal-miners did not appreciate a hot meal served at the coal face. Habit might have a big effect on food consumption. Thus much less cheese was eaten by miners in Lancashire and Durham than in other districts.

Diet of the Hospital Patient

Dr. A. LYALL reported a survey of hospital diets in Aberdeen. His method was to take an inventory of the food in selected wards. In a general hospital with 500 beds the food officially provided supplied on an average 1,680 calories, being made up of 65 g. of protein, 70 g. of fat, and 183 g. of carbohydrate daily. Food was brought in privately for the patients

to the average extent of 502 calories, making the total daily intake 2,220 calories, which was nearly identical with the estimate of 2,100 calories for the population in general. It was obvious that in the absence of private food supplies the patients would be underfed. In a maternity home the intake was 2,200 calories without private extras, and 2,540 calories with extras. Adequate feeding was essential during confinement, although overfeeding might cause obesity, particularly in multiparae. Good food in a maternity home was desirable, not only for its immediate effect, but for its educational value. In a hospital for chronic diseases the total intake was 2,130 calories.

Miss M. C. BROATCH, of the King Edward's Hospital Fund, suggested that frequent small meals might be better than fewer large meals. In school the time taken over the "Oslo dinner" had the advantage of resting the children. The goal of 1,000 calories for a school dinner seemed to be set too high, and could be reached only by the frequent inclusion of suet puddings. In hospitals monotony must be avoided at all costs. Trained food buyers and trained cooks were necessary. Equipment for cooking and conveying food was at present so bad in one large hospital that only one cooked meal a day could be managed. Miss KERLEY agreed that 1,000 calories was too high for a single meal. In meals examined in Surrey the mean protein content was 28 g., fat 25 g., and calories 668. The highest vitamin C value was 35 mg. per meal, and the mean for the year 25 mg. Dr. MAGEE, of the Ministry of Health, complained that hospitals were not procuring or applying for all the foodstuffs to which they were entitled. Hospital patients should receive 120 g. of protein daily, including 78 g. of animal protein. In one hospital no bacon, eggs, or butter were purchased. While he would not accuse those responsible of dishonesty, there was obvious room for improvement. The danger of vitamin C deficiency was slight; he had not found a single case of scurvy in 15,000 subjects examined. Dr. G. GRAHAM, of St. Bartholomew's Hospital, however, reported his recent observation of seven cases of scurvy and of hyperkeratosis of the hair follicles. He stressed the importance of vitamin C in wound healing. Prof. J. R. MARRACK spoke of the loss of appetite in workers when changed to night shift, and Miss M. ANDROSS of the inconvenience of large meals to workers operating under cramped conditions.

The last speaker was Prof. MAYNARD, of Cornell University, U.S.A., who expressed his pleasure that his official mission to this country had afforded him an opportunity to be present at the meeting and also to "look in" on the experiment on human vitamin A requirements which was being undertaken by an M.R.C. subcommittee at Sheffield. There was urgent need to convert data on food supplies and requirements into common terms that could be understood in all countries. Thus the daily requirement of vitamin A was estimated by the National Research Council of the U.S.A. as 5,000 i.u. This was intended to apply to a mixture of "preformed" vitamin A and carotene in the proportion in which they were present in most diets, and not, as was sometimes assumed in this country, to either vitamin A or carotene considered separately. There was an unfortunate tendency for lease-lend foods to change their nutritional value, at least on paper, merely by crossing the ocean! Another urgent need was for the agriculturist and the nutrition expert to co-ordinate their ideas and link up their terminology, so that food production might be harmonized with food requirements.

CONFERENCE OF PHYSIOLOGISTS IN MOSCOW

When Pavlov was still alive his pupils and fellow-workers would meet regularly at his flat, where in an atmosphere of friendliness they would discuss current work, difficult problems, achievements, new theories, and experiments. After the death of the great Russian physiologist the tradition was continued, but no longer in the teacher's home. In large halls Soviet physiologists hold conferences on physiological problems and discuss the heritage of the great scientist and his discoveries and achievements in those branches of physiology in which he did pioneer work. The ninth conference was held in 1941; the tenth opened in Moscow on March 29, 1944. In his opening address Academician Orbeli, vice-president of the Academy of Sciences and chairman of the conference, pointed out that these memorial conferences had been in abeyance on account of the war. It had become urgently necessary to evacuate laboratories into the interior. "But," he added, "there is no power on earth that can stop the onward march of the scientific thought of the Soviet people." In spite of wartime difficulties, research, far from diminishing in scope, has gone ahead into new channels. The war has created new conditions, and scientists are making full use of them. Theoretical physiology is being widely applied to problems of war medicine. The physiological causes of higher nervous activity established by Pavlov have made it possible for his pupils and research works to develop new methods for every variety of wartime trauma. Pavlov himself carried out his experiments chiefly on dogs, but now in wartime the majority of physiologists are making a study of man himself, healthy and sick, chiefly of the effects of war injuries—a study which permits of a more accurate understanding of the mechanism of higher nervous activity and offers a possibility of rendering better aid in healing the wounded.

Fifty papers were read at the conference, seventeen of them devoted to clinical physiological investigations into the effects of wartime trauma and the development of methods for their treatment, further experimental work in the study of higher nervous activity, problems of the chemical regulation of the nervous activity of the organism, and the physiology and pathology of the sense organs.

The first paper was entitled: "New Material concerning the Genesis of Old Age, and Measures for Preventing Premature Senility," written by Maria Petrova, a very close assistant of Pavlov, who spoke of her as quite the most valued and outstanding of his fellow-workers. Petrova remained in Leningrad during the whole of the siege, in spite of her advanced age—she was 70 in April. In Leningrad, or sometimes in the suburb of Koltushy, she continued her experiments on neuroses in dogs. Her paper gave the results of parts of this work. She concluded with these words: "To slow down the process of ageing is possible by intelligent management of one's life. The first principle of this intelligent management is work."

Correspondence

Work of M.R.C. Committees

SIR,—Dr. J. M. Alston's letter in your issue of May 27 about the work of committees of the Medical Research Council touches on important questions about which I may perhaps usefully make some factual comments.

The Council do not themselves claim any higher authority for the opinions of their expert committees than that stated by Dr. Alston. In science there can be no official brand of truth, and seldom any finality of judgment. Nevertheless, when it is necessary to take a decision in practical affairs this must be based on what is considered to be the best advice available at the time, although it should be realized that such advice is open to criticism by other competent and informed persons and is subject to review in the light of further knowledge.

The Council would likewise agree with Dr. Alston that their efforts in promoting and co-ordinating research are complementary to independent endeavours and work assisted from other sources. They are certainly far from wishing to exercise any restrictive function, although they must necessarily discriminate when deciding which projects should receive their positive support.

Dr. Alston is of course right in saying that new drugs were tested and evaluated before the Council set up special machinery for the purpose, but some reservations must be made. Before such machinery existed there was frequently real difficulty in securing adequate clinical trial of new remedies, especially those emanating from commercial laboratories, and in other cases the release of new remedies without any organized preliminary test had undesirable consequences. Dr. Alston is unfortunate, moreover, in his three examples of remedies alleged to have been tried without the aid of official machinery. The Medical Research Council were solely responsible for the first trials—and indeed the first production—both of insulin and of liver extract in this country. The Council also arranged clinical trials of concentrated antipneumococcal serum sent to them from America for the purpose—incidentally, Dr. Alston was a participant in their scheme. It is true that all these trials, except the later ones on pneumonia, were made before the Council had appointed a standing Therapeutic Trials Committee to assist them in the matter, but the machinery was not otherwise different.—I am, etc.,

Medical Research Council. EDWARD MELLANBY.

Aerial Convection from Smallpox Hospitals

SIR,—The article by Dr. Killick Millard (May 6, p. 628) and the letter from Prof. Picken (May 20, p. 699) raise again the difficult question of the aerial transmission of smallpox. In the 1920 Glasgow epidemic, to which Prof. Picken refers, I was unable to assure myself, after consideration of all the relevant facts, to what extent the smallpox hospital, which was then a part of Belvidere Fever Hospital, influenced the incidence round it, and concluded that the problem was left undetermined. In the latter part of the epidemic Robroyston Hospital, in a reasonably isolated situation, admitted smallpox patients, and cases did occur within a mile or two of it. But here again, as Prof. Picken says, it was impossible to exclude other possible sources.

It seemed to me that the first step to be taken was to prevent any possibility of spread from the hospital by members of the staff. Accordingly, it was arranged that the staff, including nurses, maids, and porters, should be recruited from volunteers who would undertake to live continuously in the hospital without the routine outside hours off duty, that their salaries would be increased by 50%, and that they would receive a reasonable holiday after the outbreak terminated or at intervals preceded by careful personal disinfection. This system has worked well. Since it was adopted odd cases of smallpox have been admitted, including the small outbreak in the city two years ago. Although it has not been tested out in a major outbreak, nevertheless it has given a sense of security, and I do not know of any instance where spread from the hospital, directly or indirectly, could be alleged.—I am, etc.,

Public Health Department, Glasgow A. S. M. MACGREGOR.

SIR,—Dr. Killick Millard's communication is assuredly of practical importance, for, as he says, if aerial convection can be absolved of any notable influence in the spread of smallpox public authorities will be spared much expense and their medical officers great inconvenience.

I cannot, however, think that he has been happy in selecting the Purfleet epidemic of 1901–2 as his main proof of aerial convection. Obviously if Dr. Buchanan in 1904 had to admit that even one man from the hospital ships had visited his sweetheart at Purfleet this completely invalidates the statement in his report, presumably written before this painful discovery, that "in the Purfleet epidemic we have to choose between aerial convection or nothing; there is here no question of infection, direct or mediate, through human intercourse or traffic." And, the bottom being thus knocked out of Dr. Buchanan's report, away goes the striking support that, in Dr. Killick Millard's opinon, it gives to the theory of aerial convection.

At this point I was moved to take down *Infectious Diseases*, by my old teacher and chief, that same Dr. C. B. Ker who opened the debate against aerial convection at the Epidemiological Society's meeting in 1904 mentioned by Dr. Millard, and I turned to the section on smallpox hospitals and aerial convection on page 195. The whole of it should be read, but permit me to quote some sentences which seem to be very pertinent. "What is to be the situation of the smallpox hospital? In an adequately revaccinated community this is absolutely immaterial. In Germany the very existence of such buildings is unnecessary, and stray cases of the disease are nursed in general hospitals with no ill effects." He notes that "hospital influence" is generally recognized, and gives the two theories—aerial convection and human contacts—that are held to account for this influence.

"That infection can, under certain circumstances, be carried by air it would be unreasonable to doubt. . . . It must be remembered, however, that there are more ways of leakage of infection from a smallpox hospital than by air. . . . When the Edinburgh Smallpox Hospital was in the centre of the city it was necessary, not so many years ago, to have practically a cordon of police round its walls to prevent the outside public deliberately coming into contact with the patients. And as regards a certain hospital in England, the M.O.H. found that young men from outside were carrying on illicit amours, not only with the wardmaids, but actually with girls convalescing from smallpox, and that within the hospital precincts. It is said that nowadays such breaches of discipline are impossible. It is certainly to be hoped so, but who can answer for every member of his staff?"

And after briefly recording the Edinburgh outbreak (given in greater detail by Dr. Millard in his account of the epidemiological debate), he concludes, "It is a somewhat melancholy reflection that it is only the extraordinary attitude of this country towards vaccination which renders this discussion of smallpox hospitals necessary. The whole question should be merely academic. As it is, it is unfortunately most practical." Claude Ker, as all who knew him will agree, was a man of immense sanity and rich in the uncommon virtue of common sense.

One other consideration occurs to me. Human nature is infinitely variable and the possibilities of human contacts multitudinous. Aerial convection should have no variables except the wind. If for three months the Edinburgh City Hospital could have an average of fifty smallpox patients without any evidence of infection having spread from the hospital, and this under local conditions that in England would have been illegal, is not this "negative evidence" very positive evidence indeed?

Few, I suppose, would deny the possibility of aerial convection. The problems are what is its effective range and what is its importance as compared with direct human contacts. It is, as Dr. Killick Millard says, a matter of weighing the evidence; but while sympathizing with his desire to put the matter to the test I share the dislike of Dr. Harries that the *corpora vilia* of the experiment should be the general population of a fever hospital not all protected by recent efficient vaccination. A possible, though, I admit, an unlikely, method of investigation would be in any future epidemic to surround smallpox hospitals with hutments at increasing distances from the hospital as centre, and radiating to the four cardinal points (this to test the influence of the prevailing winds); each hutment to be in a compound surrounded by barbed wire entanglements, and to be occupied by a volunteer corps of adult unvaccinated conscientious objectors.—I am, etc.,

Barrow-in-Furness. JOHN WARE.

SIR,—I think Dr. Killick Millard's views on the aerial convection of smallpox merit serious consideration by those concerned with public health teaching and practice. The evidence against this possible mode of transmission of smallpox has always lacked complete proof. If the causative organisms of enteric and dysenteric infections can be disseminated in pulverized faeces at long range during military campaigns, why should not the virus of variola, which is known to exist in the desiccated crusts of smallpox lesions, be similarly dispersed? I at any rate intend to continue telling my students about the possibility of this officially unorthodox mode of transmission until absolute proof of its invalidity is forthcoming.—I am, etc.,

Cork Street Fever Hospital, Dublin. CHRIS. J. MCSWEENEY.

Types of Diphtheria Infection

SIR,—I read with interest your annotation on misleading names in reference to *C. diphtheriae* (May 6, p. 626), and in view of the interest that this subject has for those engaged in clinical fever work I here give figures not previously published of cases of diphtheria typed by me at Knightswood Hospital, Glasgow, between Oct., 1934, and June, 1936, while acting as senior assistant to Dr. W. Dow, the then medical superintendent.

It was noted at Knightswood Hospital that the death rate for diphtheria, which had been 4.3% in 1933, had altered during the first part of 1934 to 8.2%, and that a more severe type of case was being admitted. It was decided, therefore, to type all cases of clinical diphtheria admitted, and to correlate these types with morbidity and mortality. The following were the results obtained:

Total Number of Cases—414

Type	No. of Cases	Deaths		Cardiac Complications		Paralysis	
		No.	%	No.	%	No.	%
Gravis ..	129	16	12·4	18	14	25	19·3
Intermediate	208	3	1·4	17	8·1	11	5
Mitis ..	57	—	—	—	—	1	1·7

(Also 15 atypical strain, 5 mixed. No complications or deaths.)

Classification of cases as regards severity, using the total amounts of serum given as an arbitrary measure, gave the following results:

Severity	Gravis	Intermediate	Mitis
	%	%	%
Marked	33	8	12
Moderate ..	27	31	30
Mild	40	61	58

(Mild, 0–24,000 units. Moderate, 24,000–40,000 units. Marked, 40,000 units and over.)

From the above tables it will be noted that in this series the terms gravis, intermedius, and mitis did fit into the clinical picture, although in mitis infections there was a tendency to overtreat in comparison with the other two groups, bearing out what I have since noted, that in mitis infections the local condition is often out of proportion to the general toxaemia.

In respect of gravis cases it was noted that certain strains did not produce a severe infection, and that these strains were slow in producing the typical sugar and other reactions, including haemolysis, and it was therefore thought that there was perhaps some relationsnip between the severity of a diphtheritic infection and the causal organism's haemolytic power. In an attempt to correlate these, several strains were tested for haemolytic power, using 5% suspension of human, sheep, guinea-pig, rabbit, ox, and horse corpuscles, also poured plates incorporating these bloods (guinea-pig omitted). Briefly, the conclusions come to were that intermedius strains were non-haemolytic for all corpuscles, that mitis was haemolytic, except for sheep's corpuscles and, in certain instances, ox corpuscles, while gravis could be divided into haemolytic and non-haemolytic strains, the former varying in their ability to cause haemolysis according to the corpuscles used. Gravis organisms, however, isolated from severe infections invariably caused haemolysis of human, rabbit, guinea-pig, and horse corpuscles, and certain of these also caused haemolysis of ox and sheep corpuscles when incorporated in poured plates.

These haemolytic strains appeared to be more virulent for mice than non-haemolytic strains, and the average amount of serum given in such infections was 43,000 units, as against 23,000 for non-haemolytic strains, leaving the impression that the haemolytic power of gravis strains is related in some way to virulence. It would be interesting to know the relationship, if any, between the haemolytic capabilities of gravis strains and their component *B* production as postulated by O'Meara (*J. Path. Bact.*, 1940, **51**, No. 3, 317).

As regards the response of gravis infections to ordinary antitoxin, one usually found (depending upon the day of illness) that the general toxaemia could be controlled by large doses of intravenous serum, although the local condition might progress for several days after the commencement of treatment. In recent cases one has found that adequate dosage has had to be as high as a total of 500,000 units, given over a period of several days.—I am, etc.,

J. LANDESS HORNE.
Medical Superintendent.

County I.D. Hospital, Strathpeffer.

Increase in Diarrhoeal Diseases

SIR,—The scourge of infantile diarrhoea, still prevalent in London in the early years of the present century, has been vastly diminished until these war years, campaigns against dirt, "kill that fly," etc., obviously having helped. More recent illustrations of dangers of fly-borne infections come from: (1) a cavalry camp where flies swarmed, and dysentery cases were 30 a day, rapidly reduced to *nil* by a medical officer who had flies slaughtered; (2) districts where so-called summer sickness prevails—i.e., almost universal mild or severe gastro-enteritis—always worse where hot dry weather and neglect bring swarms of house flies. The few households that escape infection are scrupulously free from fly contact. Culture plates exposed to house flies and incubated gave such illuminating results that local doctors now say, "Yes, fly sickness," when called to "summer sickness" cases, hoping thereby to impress the need for "kill that fly."

Successful breast-feeding is the best foundation for healthy life and the greatest safeguard against infant mortality, especially from infection. Infants should never be weaned in the hottest summer months. In the severe winter epidemic of infantile enteritis in London a few years ago one home had 11 babies: 4 completely breast-fed escaped infection; 7 on part or whole artificial feeding were infected and 4 of them died.

More widespread successful breast-feeding will be attained when common-sense principles—such as are applied in good dairy farms—are followed. A cow must not be chased round a field all day, must be given enough proper food and abundant water, and must be milked regularly and above all *completely*. So long as mothers are taught in maternity homes and hospitals to give "10 minutes each side" instead of to make "baby *strip* first breast before changing to second," so long will many successfully "dry themselves off" in a few weeks. Cows are specially treated to yield quality and quantity at the lowest cost; apply similar principles to human breasts for improved results to both mother and baby.—I am, etc.,

D. C. LOGAN.

London, W.1.

Osteo-arthritis after Carpal Scaphoid Fracture

SIR,—It appears from the article by Robertson and Wilkins on fracture of the carpal scaphoid, in your issue of May 20 (p. 685), that following non-union a large proportion of the cases, eighteen in all, developed varying degrees of osteo-arthritis, whereas three more were able to return to duty with no signs of arthritic changes. Why this difference? It suggests something in the individuals concerned rather than in the injury itself. Two hypotheses suggest themselves: the first, that a focus of infection was present in the individuals developing arthritis and not in the three who escaped. This explanation will not appeal to those who regard osteo-arthritis as having no infective factor. The second, that the state of the cartilage determined the issue. Some people seem to possess, perhaps by inheritance, a vulnerable form of cartilage, others not so. Subpatellar erosion is easily demonstrable and occurs in all cases of osteo-arthritis, whether the knee-joints are involved or not. It is never found in typical rheumatoid arthritis.

McCombie Young (1939)[1] found that 10% of all recruits showed subpatellar erosion. If vulnerable cartilage and a focus of infection were present this might account for the three severe cases of osteo-arthritis described.

It would be a valuable observation were a note to be made in all cases of carpal fracture of the presence or absence of patellar crepitus and of any obvious focus of infection in the teeth, posterior nares, or throat.—I am, etc.,

H. WARREN CROWE.

London, W.1.

Man's Essential Dilemma

SIR,—Your welcome notice of my *Conscience and Society* (May 20, p. 691) brings out a number of points in this book which other reviews have missed or elided. May I comment on three of these?

I do hold most firmly that Freud's major errors (whether of fact or of stress) are attributable to his own "obsessive" character, and, further, that it is to "aggressive-obsessional" characters that we owe that emotional leadership which drives us to war. But I also hold and emphasize that modern war would probably not occur if obsessional methods of thinking and feeling did not ultimately spread to *normal* men and women.

That is hardly a "narrow" conclusion. My doctrine of the universality of human prejudice and our human tendency to fall into obsessional ways of thought under stress of emotion willingly admits the political bias at once of the author and of the reviewer of this book. But no argument of mine would make "all belligerents equally guilty," any more than belief in the universality of human selfishness makes two litigants equally guilty in the eyes of a judge. Between nations, however, the trouble is to find the judge and secure the judgment. That search and attainment are among the main concerns of *Conscience and Society*.

The quotation from my concluding tract for the times: "Ten years after this present war has ceased it will no more matter who 'caused' or who 'won' it than it did ten years after the last one" requires careful reading and also the context: "Politically, mankind will be crushed and burdened

[1] *British Medical Journal*, 1939, **2**, 702.

by the problems of an aftermath. Individually, men will have forgotten all but the empty seats at table, the pensions, and occasional fireside memories. What matters about a war to the men and women concerned is the starting, the waging, and the stopping of it. Those and not the 'issues involved' are the realities of our present life." Perhaps that will all read less "curiously" in ten years' time.—I am, etc.,

RANYARD WEST.

Status of M.R.C.P.s

SIR,—The Royal College of Physicians of London has recently published a report on medical education, and I desire to point out an anomaly and what I believe to be a disadvantage in the work of the College. Members play no part. Members, who hold a high and valuable diploma, have never held a statutory position in their College. I believe it is high time that members should be given a better position in the Royal College of Physicians and a share in its government. They could, if allowed, contribute substantial and valuable material to the work of the College. There are almost exactly a thousand members of over ten years' standing, but all are rigidly excluded from any share in the activities of the College, and under the existing constitution the great majority are permanently excluded.—I am, etc.,

Peterborough. G. F. WALKER.

Medical Education: Principles and Method

SIR,—One of the more amusing aspects about the present discussions on medical education is the disgusted contempt with which the teachers seem to regard the average student. It would appear that he is an ignorant, uncultured fellow, hardly fit to be received in educated society, and certainly in no state to profit from the pearls of wisdom which fall from the lips of the teachers. Boys, one expects them to say, were very different in *their* young days. It rather looks as if science has displaced the unfortunate classics in yet another way; instead of: "Why! the boy can't even compose a decent hexameter!" it is now, "Good heavens! the young nincompoop has no conception of modern philosophical theory!"

Dr. Walshe complains that he has yet to meet a student "who has a clear idea of what is meant by a natural law or how it is defined, and who realizes that all science comes from the meeting of the observational and conceptual orders of experience. . . ." I can well believe it. I doubt whether even Dr. Walshe talked like that at the age of 18 or 20. At that age a boy is learning how to *do* things, not how to talk about them in high-sounding phrases. It is reality he is after, not abstractions; those will come later of themselves if he is of that turn of mind; if he is not, I do not think anything will make him so.

It would, of course, be quite easy to produce students who could recite stuff about conceptual orders of experience *ad lib.* Very nice, too, and most useful training for a philosophical lectureship; but not really of much guidance when it comes to Mrs. So-and-so's urgent post-partum haemorrhage, which is the sort of problem the students are really being trained to tackle. What we must bring ourselves to realize, painful though it may be, is that the great majority of medical students are apprentices to a craft; as G.P.s they will be craftsmen whose job, as Sir Thomas Lewis has pointed out, is to apply what they have been taught according to established principles, not to experiment and theorize. You do not expect the man who repairs your car to be familiar with Fourier's "Theory of Heat"; you expect him to listen to the engine and tell you whether that funny noise is really a "big-end" gone or just imagination. You do not even expect him to know much about the "general principles of scientific thought." You do hope he is intimately acquainted with the ailments of motor cars and how to cure them.

It may be argued that this is too narrow a view; that even the motor mechanic would be all the better if he were a bit of a scientist as well; *as well*—not instead. But art is long and life is short. When a practical job has to be done the "how-to-do-it" knowledge is what counts. The reason why, and the reason behind that, and so on in an infinite series, are no help, except in so far as the first few terms may serve as aids to memory. Unfortunately, medicine consists largely of unrelated facts, and so long as that remains true students will just have to commit these facts to memory as best they can.

Not that I have any wish to belittle science or even its more abstract principles. But I suggest there is some danger of overestimating their importance in practical medicine. It is apparently possible to be a very efficient doctor with only the crudest notions of elementary physics, for example. I remember a student being asked by an eminent honorary why the mediastinum moves over towards a collapsed lung. The student thought it was due to a pressure difference: the pressure on the sound side pushed it over. "Pressure?" said the honorary. "Pressure has nothing to do with it; it's pulled over by suction!" There was no "come-back" to that one, of course, and the teacher moved serenely on to the next bed, followed by the admiring crowd of students, who would cheerfully have said it was pulled over by the moon if they thought that was what a potential examiner wanted them to say. Quite right too; only a research worker wants to know why; sufficient for the practical man that it is so.

There is another objection to the attempt to teach principles before practice (or another aspect of the objection), and that is that the human mind does not work that way—at least, not if it can help it. You do not try to teach a child the philosophical principles of mathematics before teaching it to add and subtract, although in logic they come first. The results would be sadly disappointing if you did. The facts must be demonstrated and the techniques learnt first; philosophizing comes later. That is what I meant by saying that the preclinical student is still learning to *do* things: he is still learning (and re-learning) how to measure, and weigh, and precipitate, and connect batteries to induction coils (lots of them can't do that), and a host of other strange things which in after-years will become second nature. He might be expected to infer something of scientific method from all this, and so, I think, he does, according to his lights and interests. A really good teacher could help him to infer more, but teachers as good as that are born, not made.

The point I am trying to make is that the vision of graduates issuing forth from their medical school equipped with a mature scientific philosophy, not to mention an ability to behave with common sense on all occasions, is a Utopian dream. The real scientific mind is a rarity, and it starts to be one in early childhood. If you spot one, just encourage it and give it scope; it will teach itself. Moreover, if you give it half a chance it will not go into general practice. But the ordinary people, who are going to make ordinary efficient medical craftsmen in time, must be taught their trade by means appropriate to the object *they* have in view.—I am, etc.,

Brancepeth, Co. Durham. W. E. HICK.

SIR,—Dr. Walshe seems rather exacting. Why should a clinical clerk know what is meant by "the structure of experience"? The Church implants real grace in the young not by teaching theology but by using perceptual myths. "For effective teaching there must be the disk of really apprehended dogma; rays diversely reflected and refracted from clouded sources will not do." To know "the direction in which to look for light" the student must memorize facts, must be taught to perceive, must do routine techniques, and must be given a framework of crude "Platonic" concepts—such as the textbook diseases and concepts implied by a method of case-taking—to act as his "organs of interpretation" akin to his organs of sense. If at first he imagines given symbols to be adequate reality-references he can be undeceived later.

Minds capable of continued reflection are a minority; the majority want definite snippets of experience. Also generalization of process-likeness is a talent not always found in the man who is apt to discriminate differences in percepts. Often what delights the synthesizing philosophic mind fatigues and irritates the analytic technique-loving mind; often it exasperates the philosopher when the analytic mind confidently applies to a whole his logical deductions from an abstracted past. A doctor who considers all statistical results and all pharmacological tests superior to clinical judgments is no wiser than the man who yells "Equal pay for equal work" and forgets to add "by equal people."

A good teacher saves a student much labour, but students should rightly learn many more facts than they need in practice

routine. It is the physiology that a man has forgotten that enables him to read, e.g., Burridge and Adrian, with some ease, understanding, and profit.—I am, etc.,

Greenford. W. THOMSON BROWN.

SIR,—Dr. Walshe (May 27, p. 728) rightly emphasizes the necessity for teaching the medical student the principles involved in scientific thought and method. It would be a great help to the development of the student's intellectual powers if he were also taught that these principles could be applied to other problems in life besides medicine. Doctors are too often swayed by emotion rather than guided by reason when discussing non-medical subjects.

Another much-needed reform is the appointment of a general practitioner to the staff of every teaching hospital, for most of the students take up general practice after they have qualified. The idea of a G.P. let loose among students who are being carefully nursed along academic lines appals the average teaching consultant, but there could be no harm in having a G.P. present at a medical round once a week to add to the discussion of the cases.—I am, etc.,

London, W.8. H. STEPHEN PASMORE.

Medical Certificates for Special Rations

SIR,—It is with appreciation and relief I see the signs of a small revolution by the Guildford Practitioners' Group (May 20, p. 701) regarding medical certificates for special rations. May I also add my little voice and hope that other practitioners and branches of the B.M.A. will take this matter up.

My main complaint, and it is made with considerable feeling, is the issuing of the following certificates for extra milk and eggs for active tuberculosis of all types and for gastric, duodenal, and anastomotic ulcers. Now, Sir, whether the war lasts another week or another generation these people will still have their gastric ulcers and active tuberculosis, and I fail to see why we should be compelled by the Ministry of Food to issue continuous certificates every three months or less for these patients. Even if their ulcers cause them no more pain, with the present difficulty in getting milk the patients will always ask for their milk certificate, and the doctor must therefore give it to them.

There is also another point against which I should like to see the medical profession protest, and that is the Board of Trade's request for certificates for corsets and brassières. Under present emergency conditions I have no objection, and I am sure that members of the medical profession will agree with me, to the initial certificate requesting the manufacture of the article required. What I do object to is that my obese, visceroptotic, and post-maternity patients, etc., should stand up in front of me while I gaze upon their garment and see whether it fits. I am no corset or brassière expert: I do not know a doctor who is; and, furthermore, if the garment does not fit, then who knows better than the patient? Over the last six months I have refused to sign any certificates stating that the particular garment is to my satisfaction for the complaint for which it is issued. The manufacturers concerned are persistently writing or 'phoning me, and I am at present awaiting a call from a representative of the Board of Trade.

I feel that the only way in which we can stop this unnecessary waste of time and paper is for individual Branches to make the complaint, and I am sure that if there are enough complaints the B.M.A. will have to do something.—I am, etc.,

Rugby. T. E. WAINE.

The Colonial Medical Service

SIR,—To Sir Philip Manson-Bahr's tribute (May 27, p. 732) to the work of members of the Colonial Medical Service, which I heartily endorse, I could add more: But I will only mention two other examples: one that of Dr. J. W. Field, Chief Malaria Officer at the Institute for Medical Research of Malaya (with whom I had the honour to work); and the other that of Dr. Gordon Ryrie, Superintendent of the Leper Settlement at Sungei Buloh, F.M.S. In 1937 Dr. Field refused promotion rather than discontinue his work on the chemoprophylaxis of malaria. Dr. Ryrie's work for the lepers brought out whatever latent happiness a leper colony could enjoy, and brought such satisfaction to himself that in 1941–2 he preferred to remain under the Japanese invaders rather than escape and leave the colony.

If those diehards of the medical profession who consider that the fee is the only or the main incentive to good work could once free themselves from their chains, perhaps they too would find such satisfaction in their work itself that memories of the "money incentive" would become as a bad dream that is passed.—I am, etc.,

London, W.1. COLUMBA GUEST.

SIR,—From time to time you print an advertisement for medical officers for the Colonial Medical Service. May I draw attention to two statements in it which are misleading: (1) "There are ample opportunities for work in medical research." (2) "There are large numbers of superscale posts to which promotion is on merit." Any chance of work in medical research is eliminated by a reduced staff and enormously increased routine clinical pathology and medico-legal work. Promotion is usually on seniority; probably rightly so, as it at least eliminates any sense of favouritism.

I do not wish to put men off the Colonial Medical Service. I think it is good. But I feel they should not apply for a post on the strength of an ingeniously worded advertisement.—I am, etc.,

R. D. REID,
Pathologist, Gold Coast.

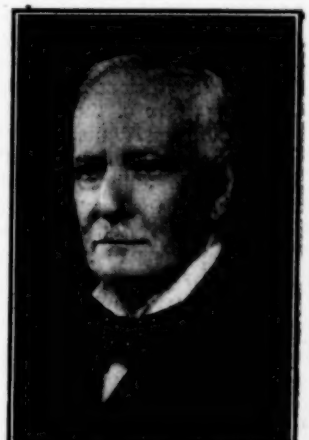
Obituary

SIR CUTHBERT WALLACE, BT., K.C.M.G., C.B.

Former President, Royal College of Surgeons

Sir Cuthbert Wallace, who died on May 24, served his profession in many high offices, and will be remembered with warm regard and admiration by his colleagues and a host of former pupils.

Cuthbert Sidney Wallace was born in 1869 at Surbiton, Surrey, the son of the Rev. John Wallace. He was educated at Haileybury College, a school in which he continued to take the greatest interest throughout his life, serving for many years on its Council. From Haileybury he went to St. Thomas's Hospital, and qualified in medicine in 1891. His bent was towards obstetrics and surgery, and he became a Fellow of the Royal College of Surgeons of England within two years of his qualification, and in the following year, 1894, M.B., B.S.Lond., obtaining the gold medal in obstetric medicine and qualifying for the gold medal in surgery.



[*Lafayette*

St. Thomas's was one of the great interests of his life. He served it as surgeon for many years, and on reaching the age of retirement was elected consulting surgeon. He was also lecturer on surgery in the St. Thomas's Medical School and afterwards Dean. Both as surgeon and as teacher, and in everything to do with the School, his skill and wisdom and devotion laid it under a great debt, and generations of St. Thomas's men in all parts of the world have occasion to think of him with affection and gratitude. He had also been surgeon to the East London Hospital for Children.

One of the outstanding experiences in his early life was his service in the South African War. In the autumn of 1899 he was asked by his friend Sir Anthony Bowlby (who, like himself, was to become President of the Royal College of Surgeons) to join the Portland Hospital in South Africa, and, Wallace eagerly assenting, they worked together there until the middle of 1900, when the hospital was dismantled. On their

return the two surgeons collaborated in producing a small book relating the story of the Portland Hospital. That, however, was only the prelude to a much more exacting and prolonged military experience. During the war of 1914–18 Cuthbert Wallace was attached as consulting surgeon to the First Army of the British Expeditionary Force and eventually became Major-General, A.M.S. Here again he was in close association with Bowlby, who, although working with the Second Army, was attached to headquarters at St. Omer, and had oversight of both First and Second. Wallace's work for the Forces during that war earned him widespread recognition. It was characterized by the thoroughness and care for detail which marked all that he did. He was mentioned in dispatches and received the C.M.G. in 1916 and the C.B. (Military) in 1918, and at the end of the war he was advanced to K.C.M.G. He wrote in collaboration with Sir John Fraser a small volume entitled *Surgery at a Casualty Clearing Station*, embodying the result of their joint experiences in that type of work during several years on the Western Front, and containing much of value for the next generation of war surgeons who came along twenty years later. It was illustrated by many sketches by Lady Fraser. When, in April, 1919, the British Medical Association held a special clinical and scientific meeting to review the medical lessons of the war he acted as joint general secretary.

For very many years Cuthbert Wallace was closely identified with the work of the Royal College of Surgeons of England. He was elected to its Council in 1919 and served until 1935, when he was made President, occupying the chair for three years. He had been a member of the Board of Examiners from 1919 to 1929, Bradshaw Lecturer in 1927, and Hunterian Orator in 1934. Many other useful activities occupied him during the later years of his life when he no longer worked in the operating theatre. Outstanding among them was his directorship of the medical services of Mount Vernon Hospital and the Radium Institute. He was also a member of the Radium Commission. He interested himself greatly in the work of the British Empire Cancer Campaign and was chairman of some of its committees. He served for some time on the Medical Research Council and was a valued member of the Army Advisory Council. His interest in the day-to-day work and difficulties of the profession showed itself in his presidency of the London and Counties Medical Protection Society. He was not content to appear at the annual meetings and make a brief speech, but he attended regularly the council and committee meetings and maintained a real interest in the detailed work of the society. As a chairman he was most businesslike. No time was wasted. The presence in the chair of this quietly efficient and dignified man made everyone feel that they must give of their best.

Sir Cuthbert Wallace's published works include volumes on the war surgery of the abdomen and on prostatic enlargement. He was the author of many contributions to *St. Thomas's Hospital Reports* and the *Transactions* of the Clinical and Pathological Societies. He was also one of the editors of the Surgery Section of Sir William Macpherson's *Medical Services of the War* (1914–18).

He was made a baronet in 1937. He held honorary doctorates of Oxford, Durham, and Birmingham, was an Honorary Fellow of the American Surgical Association, and an Officer of the Legion of Honour. He was also a J.P. for the County of London.

Sir FRANCIS FRASER, Director-General, Emergency Medical Service, writes:

Cuthbert Wallace disliked strongly the idea that he might have to give in before the end of the war. A few days before his death he was planning how he would still be able to help with advice, even though he could no longer attend the conferences at the Ministry of Health. For nearly five years as Consultant Adviser in Surgery to the Emergency Medical Service he served the country well and gave freely from his store of experience of surgery in war and in peace, from his knowledge of men, and from his capacity for wise and balanced judgment. In the earlier years of the war he visited most of the larger hospitals in the South of England and reported on their strong points and their weaknesses and the part each should play in the Emergency Scheme. Nothing he said was taken amiss: it was so honest and so clearly helpful. The difficulties and discomforts of travelling kept him at home for the past year or more, and it was as Chairman of the Consultant Advisers that he was able to continue to help so well. Many problems arise when civilian hospitals and civilian doctors are required to deal with Service patients, and the more difficult of these were kept for discussion with Wallace. It is not, however, for his help in solving administrative difficulties that we at the headquarters of the E.M.S. owe so much to him. It is for his loyal support at all times, for his gentleness, for his friendliness, and for the atmosphere of goodness that he brought with him.

Major-General Sir ERNEST COWELL, K.B.E., C.B., D.S.O., writes:

May I add my tribute to the honoured and beloved memory of Sir Cuthbert S. Wallace. As surgical specialist to 23 C.C.S. and later O.C. 1 C.C.S. in France 1915–19 I came into intimate contact with Sir Cuthbert, who was Consulting Surgeon to the First Army. His advice and judgment were sound and always helpful. He never issued surgical directives but rather "suggestives," explaining the general idea and leaving details to the individual surgeon. His work on military abdominal surgery will be remembered, and his dictum, "The surgeon who does not trust the peritoneum is not fit to do abdominals," is still true to-day. Sir Cuthbert was an ideal Army surgeon, unselfish, devoted, courteous, and thoughtful. His name deserves undying fame.

By the death of Dr. ROWLAND HAMER at Ashton-under-Lyne on May 25, in his 70th year, the area has lost a physician who, by his original and striking personality, had earned the respect and warm regard of all his colleagues and the gratitude of many patients. He combined practical sympathy for the more needy of his clients with a sound clinical judgment, and leaves behind a well-deserved reputation as a doctor of the old school. He graduated M.B., Ch.B. at Manchester in 1899, served in France and Belgium during the last war, and had held office as a member of the Ashton-under-Lyne Town Council.

The following well-known medical men have died abroad: Dr. R. O. Rosenberger, professor of immunology and bacteriology at the Medical College of Philadelphia, aged 64; Geh. Med. Rat. Prof. Carl Jacobj, emeritus professor of pharmacology at Tübingen and one of Schmiedeberg's pupils, aged 87; Prof. Alfred Kleinknecht, for many years leading surgeon at Mulhausen, Alsace, aged 73.

Universities and Colleges

UNIVERSITY OF EDINBURGH

Sir John Fraser, Bt., K.C.V.O., honorary surgeon in Scotland to H.M. the King and regius professor of clinical surgery in the University, has been elected by the curators of Edinburgh University to the office of Principal, and will take up the duties of that post on Oct. 1.

ROYAL COLLEGE OF PHYSICIANS OF EDINBURGH

Mr. Norman M. Dott, F.R.C.S.Ed., will deliver a Morison Lecture in the Hall of the Royal College of Physicians, 9, Queen Street, Edinburgh, on Friday, June 23, at 5 o'clock on "Experiences in Neuropsychiatry, Edinburgh, 1940–4."

The Services

Temp. Surg. Lieut. T. Haw, R.N.V.R., has been mentioned in dispatches. This name appears in a list of awards for outstanding courage, determination, or devotion to duty in certain of H.M. ships in successfully escorting convoys to North Russia in the face of attacks from enemy submarines and aircraft.

CASUALTIES IN THE MEDICAL SERVICES

Died of wounds in Italy.—Capt. G. E. Yardley, R.A.M.C.

Killed.—War Subs. Capt. N. W. Jones, Temp. Major R. Stuppel, R.A.M.C.

Wounded.—Temp. Major G. S. Ferraby; War Subs. Capts. A. P. D. Montgomery, C. P. Silver, and A. Wattison, R.A.M.C.

Medical Notes in Parliament

New Health Service: Medical Man-power

On May 23 Mr. MESSER asked the Minister of Health how many consultants were at present available in Great Britain and Northern Ireland, and how many he estimated would be required for domiciliary and hospital work in the proposed National Health Service. Miss HORSBRUGH said she could only answer for England and Wales. Detailed information was not available, but it was hoped that considerable light would be thrown on the present supply and future demand by the surveys mentioned in the reply given to Sir Douglas Hacking on Feb. 3.

Dr. RUSSELL THOMAS asked on May 25 how many doctors would be required to undertake the proposed National Health Service. Mr. WILLINK said there was no reliable means of assessing this at present, but he had no reason to doubt that, when doctors returned from the Forces, the number available would suffice to enable the Government to make a good start with the new health service.

New Health Service: "State-salaried Officials"

Dr. RUSSELL THOMAS on May 25 further asked Mr. Willink how many doctors he intended to make State-salaried officials on the initiation of the proposed National Health Service. Mr. WILLINK said he could not accept the implication of the question. There was no intention of turning doctors into State-salaried officials. Even where—as in the Health Centres—remuneration by salary was mentioned it was suggested as one among other possible alternatives.

Medical Supplies for China

On May 23 Mr. EDEN, replying to Mr. A. Edwards, stated that public subscriptions in this country had been devoted directly to relieving the distress in China arising out of the war. Subscriptions to the United Aid to China Fund totalled £1,122,000, of which £930,000 had been remitted to China for distribution by Madame Chiang Kai-shek. This included medical supplies to the value of over £6,500. The expenditure of the war organizations of the British Red Cross and Order of St. John to date, with commitments, amounted to £524,100, including an allocation of £100,000 for the purchase of medical supplies and drugs. Under this head some £25,000 had already been sent and the balance was in course of supply and transport. Over £200,000 was raised by the former British Fund for the Relief of Distress in China and distributed there on relief work and the purchase of medical and hospital supplies.

Smallpox at Shaftesbury

On May 25 Mr. ALFRED EDWARDS asked if the Minister of Health had considered the complaint from the medical officer of health at Shaftesbury, and if he had any further statement to make on the recent case in that town which was diagnosed as smallpox.

Mr. WILLINK replied that when he stated in a previous answer that the diagnosis of smallpox had not been confirmed he meant to convey no more than that the diagnosis was not free from doubt. He was sorry that this expression had lent itself to misinterpretation. He intended no reflection on the clinical skill and judgment of the medical officers who held the case to be one of smallpox. The steps they took with admirable promptitude to prevent the spread of infection were entirely right. He was communicating with them in this sense. The case had been properly recorded by the Registrar-General as one of smallpox.

Diphtheria in Germany.—On May 23 Mr. VIANT asked the Minister of Health whether he had any information about the incidence of diphtheria in Germany during the past winter. Mr. WILLINK said that the only information he had at present related to periods of five consecutive weeks in October and November last, one week in December, and one in February. In those seven weeks there was a total of 56,658 recorded cases of diphtheria in Germany.

Women can now have training at Government expense and receive wages during training in domestic work. This is the latest step towards providing hospitals and institutions with the domestic workers they need. The scheme of training has been based on recommendations of the Standing Advisory Committee on Institutional Domestic Employment which was set up by the Minister of Labour and National Service after his acceptance of the Hetherington report as a basis for the supply of labour, and is part of his plan for raising the status of the domestic worker generally. Courses can at present be provided in only a few areas. Mobile women taking training away from their home areas will be billeted. Immobile women can only be accepted if there is a course in their area.

Medical News

The annual general meeting of the Royal Medical Benevolent Fund was held at the Royal Society of Medicine on May 25, when the balance sheet and accounts were presented and adopted. The resignation of the honorary treasurer, Dr. Lewis G. Glover, was received with great regret and Dr. C. Luther Batteson was elected in his place. New members of the committee are: Lord Moran, Mr. Eardley Holland, Sir Stanley Woodwark, Mr. V. Zachary Cope, and Dr. C. M. Hinds-Howell.

At a meeting of the Council of the Imperial Cancer Research Fund held on June 1 Prof. H. R. Dean was re-elected chairman of the Council for the ensuing year and Sir Hugh Lett, Bt., was re-elected vice-chairman. The Elizabeth Wills Allen Fellow, Dr. L. Foulds, and the Alice Memorial Fellow, Dr. R. J. Ludford, were both re-elected to their fellowships. The Council received with gratitude, from Messrs. Townson and Mercer, a gift of scientific apparatus to the value of £250.

Dr. W. S. Maclay will give a lecture on "Neuropsychiatry in Wartime" to the Association of Austrian Doctors in Great Britain to-morrow (Sunday, June 11), at 11 a.m., at 69, Eton Avenue, N.W.3 (near Swiss Cottage Station). Guests are welcome.

At a sessional meeting of the Royal Sanitary Institute in Bolton Town Hall on Saturday, June 17, a paper on the functions of nurseries in peacetime will be read by Dr. R. M. Galloway at 10.15 a.m. The meeting arranged for Saturday, June 24, at Haywards Heath has been postponed in view of the present situation.

The next meeting of the Biochemical Society will be held in the Physiology Department, University College, Dundee, on Saturday, June 17, beginning at 2 p.m.

The new mass miniature radiography centre for the City of Nottingham began work on May 17 under the supervision of Dr. A. E. Beynon. The centre is staffed by a team of three radiographers, an organizing secretary, four clerks, and a woman marshal.

On May 21 at Great Missenden Parish Church a handsome reredos was dedicated to the memory of Edgar Olive Turner, M.B. Dr. Turner, who died some two years ago, had practised in Great Missenden and district for nearly half a century and was much beloved by the community. He was at one time a member of the Council of the B.M.A.

The special meeting of the Council of the Medical Women's Federation arranged for July 8 to discuss the White Paper on a National Health Service has been postponed indefinitely, in response to the Government's request that conferences be cancelled.

A meeting of the executive committee of the Cancer Control Organization for Edinburgh and South-East Scotland, the objects of which include the investigation, care, and cure of cancer, was held in Edinburgh on May 3. Among the subjects discussed were the part the organization might play in the future cancer service, the publication by the organization of a volume consisting of studies on mammary cancer, and the fuller use by medical practitioners of the system of the organization whereby practitioners may arrange for the issue of travel vouchers to enable persons suffering, or suspected of suffering, from malignant disease to attend at Edinburgh Royal Infirmary for treatment or for examination free of charge. The honorary treasurer of the Cancer Control Organization is Prof. William Annan, C.A., 22, Charlotte Square, Edinburgh, to whom subscriptions, donations, or bequests by way of legacy should be sent.

The King has granted permission for the wearing of the insignia of Commander of the Order of St. Olav conferred by the King of Norway upon Mr. Charles Alfred Keogh, F.R.C.S., in recognition of services rendered by him during the war.

The British Pharmacopoeia Commission announces that the period during which ointments prepared according to the original formulae of the *B.P.*, 1932, or addenda, may be dispensed or supplied is extended until further notice.

The Haldane Silver Medal and Prize has been awarded by the Institute of Public Administration for an essay entitled "Child Neglect: a Problem of Social Administration," written by Dr. A. E. Martin, assistant M.O.H. for Leicestershire and M.O.H. for the Wigston and Oadby Urban Districts.

The name of A. C. Kirton, M.B., ship surgeon, appears in a list of names published by the *London Gazette* as commended for brave conduct in the Merchant Navy, when their ships encountered enemy ships, submarines, aircraft, or mines.

The William Osler Medal of the American Society of the History of Medicine has been awarded to George Edward Murphy of the Medical School of the University of Pennsylvania for his study entitled "The Evolution of our Knowledge of Rheumatic Fever."

The April number of the *Bulletin* of the National Association for the Prevention of Tuberculosis (Tavistock House North, Tavistock Square, W.C.1) announces that the N.A.P.T. is to undertake a comprehensive investigation into the psychology of tuberculosis, to last in the first place for some twelve months. All types of patients with pulmonary tuberculosis will be studied. The work will be carried out by a competent psychiatrist with an assistant psychiatric social worker. Full personality studies will form the basis of the work.

The Minister of Health has been in consultation with the Minister of Labour and National Service and the Minister of Pensions about questions which arise in connexion with the placing in suitable employment of persons suffering from some form of psychoneurosis or psychosis. Such persons often break down in the employment in which they have been placed and the Minister of Labour has experienced difficulty in finding them work which they can usefully perform. The Ministry of Labour and National Service consider that the advice of a skilled psychiatrist would be most helpful to officers at Employment Exchanges in determining whether a worker known to be suffering from psychoneurosis or psychosis whom it has hitherto been difficult to place in industry is, in fact, suitable for employment and, if so, what type of employment he or she is best fitted to undertake. Arrangements have accordingly been agreed between the Departments concerned.

The York Clinic for Psychological Medicine at Guy's Hospital, after serving some of the general needs of the hospital since the "blitz" of 1940–1, has now been opened for its original purpose. Thirty of the forty-three beds are reserved by the Emergency Medical Service for psychiatric casualties among officers of each of the Services; the remainder are available for civilians. The acting medical director is Dr. Felix Brown and the sister-in-charge is Miss E. Purcell.

EPIDEMIOLOGICAL NOTES

Discussion of Table

In *England and Wales* notifications of measles, acute pneumonia, and whooping-cough exceeded those of the previous week by 123, 102, and 71 respectively, but those of scarlet fever were lower by 58.

Lancashire reported a decrease of 71, and Yorks West Riding 47, in the notifications of scarlet fever, the total for pneumonia, however, going up by 23 in the latter county. The incidence of whooping-cough rose in Kent and Middlesex by 48 and 35 respectively. Essex reported 48 more cases of measles than last week, but in contrast to the general trend Kent reported 69 cases fewer, and Durham 53. The incidence of diphtheria in Lancashire and Yorks West Riding remains relatively high, one-third of the total cases for the country being notified in these counties during the week.

Dysentery is still prevalent, and 276 cases (4 fewer than in the preceding week) were recorded. The largest of the new outbreaks was Yorks East Riding, Bridlington M.B. 37. The other principal centres of infection were Lancashire 53, Kent 24, London 18, Northamptonshire 17, Southampton 13, Derbyshire 13, Warwickshire 12.

In *Scotland* notifications for measles were 81 fewer than last week, scarlet fever 27, and diphtheria 23, but the total for dysentery rose by 27. The 138 cases of diphtheria were the fewest reported for five months. The largest returns for dysentery were Edinburgh 21, Lanark County 17, and Glasgow 10.

In *Eire* the only change in the trend of infectious disease was an increase of 50 in the notifications of measles, almost one-third of the total being notified in Dublin C.B. A case of typhus was reported from Roscommon, Castlerea R.D.

In *Northern Ireland* there were 22 more cases of diphtheria than last week, almost half the total being notified in Londonderry C.B. Slightly more than half the cases of scarlet fever were notified in Belfast C.B.

Week Ending May 27

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 1,853, whooping-cough 2,427, diphtheria 507, measles 2,662, acute pneumonia 774, cerebrospinal fever 57, dysentery 274, paratyphoid 4, typhoid 9.

Eire: Births and Deaths Week Ending May 13

The figures for births and deaths for Eire were not received in time for publication in the general table. The figures were: births 593, deaths 222, deaths 0–1 year 29; death rate 14.4; deaths from paratyphoid (B) 1; scarlet fever 0; whooping-cough 0; diphtheria 1; measles 2; diarrhoea 8; influenza 1; pneumonia 9.

No. 20

INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a summary of Infectious Diseases and Vital Statistics in the British Isles during the week ended May 20.

Figures of Principal Notifiable Diseases for the week and those for the corresponding week last year, for: (a) England and Wales (London included). (b) London (administrative county). (c) Scotland. (d) Eire. (e) Northern Ireland.

Figures of Births and Deaths, and of Deaths recorded under each infectious disease, are for: (a) The 126 great towns in England and Wales (including London). (b) London (administrative county). (c) The 16 principal towns in Scotland. (d) The 13 principal towns in Eire. (e) The 10 principal towns in Northern Ireland.

A dash — denotes no cases; a blank space denotes disease not notifiable or no return available.

Disease	1944					1943 (Corresponding Week)				
	(a)	(b)	(c)	(d)	(e)	(a)	(b)	(c)	(d)	(e)
Cerebrospinal fever	62	6	20	4	3	68	2	19	2	2
Deaths		1	2				2	1		
Diphtheria	598	23	138	75	55	619	41	162	65	19
Deaths	3	1	2	1	—	12	2	1	1	—
Dysentery	276	18	81	—	—	146	18	73	—	—
Deaths				—	—				—	—
Encephalitis lethargica, acute	—	—	—	—	—	—	—	—	1	—
Deaths		—					—			
Erysipelas			44	8	3			42	9	1
Deaths		—					—			
Infective enteritis or diarrhoea under 2 years				17					13	
Deaths	45	9	14	8	4	63	16	6	9	3
Measles	2,255	213	479	211	25	8,377	430	561	17	33
Deaths	1	—	—	2	—	7	1	3	—	—
Ophthalmia neonatorum	98	6	35	—		97	5	17	—	—
Deaths										
Paratyphoid fever	3	—	9 (B)	16 (B)	—	4	—	1	—	—
Deaths	—	—	—	1 (B)	—	—	—	—	—	—
Pneumonia, influenzal*	806	63	22	8	8	672	38	4	—	3
Deaths (from influenza)	17	2	2	1	—	15	3	1	1	—
Pneumonia, primary			205	35				208	23	
Deaths		30		9	6		26		13	12
Polio-encephalitis, acute	1	—				—	—			
Deaths		—					—			
Poliomyelitis, acute	5	1	2	—		7	—	1	3	—
Deaths		—					—			
Puerperal fever		1	12				2	15		—
Deaths							—			
Puerperal pyrexia†	142	4	16	1	1	183	7	18	1	7
Deaths		—								
Relapsing fever	—	—			—	—	—			—
Deaths										
Scarlet fever	1,733	121	172	20	56	1,740	155	250	49	41
Deaths	2	1	—	—	—	—	—	—	—	—
Smallpox	—	—	—	—	—	—	—	—	—	—
Deaths				—	—				—	—
Typhoid fever	5	—	2	3	1	12	1	2	15	1
Deaths	—	—	—	—	—	—	—	—	—	—
Typhus fever	—	—	—	1	—	—	—	—	—	—
Deaths				—	—				—	—
Whooping-cough	2,619	239	182	38	25	2,446	128	287	14	36
Deaths	11	4	1	—	1	15	1	4	1	1
Deaths (0–1 year)	324	46	79	29	18	375	53	63	46	27
Infant mortality rate (per 1,000 live births)										
Deaths (excluding still-births)	4,077	634	584	222	136	4,454	637	627	242	143
Annual death rate (per 1,000 persons living)			13·4	14·4	‡			14·1	15·9	‡
Live births	7,339	882	1,030	593	350	6,985	817	1,037	389	345
Annual rate per 1,000 persons living			20·9	—	‡			21·2	25·6	‡
Stillbirths	222	25	30			221	18	34		
Rate per 1,000 total births (including stillborn)			28					32		

* Includes primary form for England and Wales, London (administrative county), and Northern Ireland.

† Includes puerperal fever for England and Wales and Eire.

‡ Owing to evacuation schemes and other movements of population, birth and death rates for Northern Ireland are no longer available.

Letters, Notes, and Answers

All communications with regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1. TELEPHONE: EUSTON 2111. TELEGRAMS: *Aitiology Westcent, London.* ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.

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ANY QUESTIONS?

Tobacco, Alcohol, and Hypertension

Q.—*What part, if any, is played by tobacco and alcohol in the production or maintenance of so-called essential hypertension? Many intelligent patients with this condition, and otherwise fit, have asked me what are the scientific reasons that prompt me to advise them to abstain from these drugs. What change in the B.P. readings is likely to occur in an otherwise fit man who is a heavy smoker and moderate drinker with a B.P. of, say, 165/110 if he completely abstains from these consolations for some months?*

A.—There is no evidence that tobacco or alcohol plays any part in the production or maintenance of essential hypertension, and the patient under discussion is unlikely to show any improvement in his blood pressure if he abstains from them. It is true that transient rises in blood pressure have been described following smoking, particularly in those unaccustomed to tobacco and in individuals with effort syndrome or hyper-reactive vascular systems. These effects are apparently mediated by peripheral vasoconstriction of nervous origin and are quite different from the humoral vasoconstriction of essential hypertension. There is no evidence that the blood pressure is higher in smokers than in non-smokers, or that smoking has any uniform effect on the blood pressure of hypertensives. As emotional factors are believed to be important in maintaining hypertension, it would seem undesirable to put the patient to the strain of giving up tobacco entirely. On the other hand, both scientific and clinical evidence indicate that tobacco is harmful in Buerger's disease, in endarteritis obliterans, and in angina pectoris. In alcoholism the tendency is to low rather than high pressure. The patient should be told that moderation in all things is the golden rule in the treatment of hypertension, and he should practise a temperance which implies neither complete abstinence nor excessive indulgence in tobacco and alcohol.

How to Interpret the W.R.

Q.—*To what extent does a positive Wassermann reaction or similar test indicate the presence of syphilis? If the test is positive when repeated, is one justified in starting treatment? How should one interpret (a) a weak positive, (b) a doubtful Wassermann reaction? To what extent does a negative reaction exclude syphilis?*

A.—A positive W.R. carried out by a reliable pathologist is confirmatory evidence of syphilis if a patient has clear-cut signs of the disease. *In the absence of signs of syphilis no positive serum test, no matter by whom it is done, even if it is positive on a second specimen, in itself justifies institution of antisyphilitic treatment.* Quite a number of pathological conditions—mostly tropical diseases and therefore not likely to be met with in this country—may be responsible for false positive reactions. But such conditions as glandular fever, scarlet fever, pneumonia, late tuberculosis, diabetes mellitus, enteric fever, and malignant tumour—to mention only a few—have been incriminated from time to time. Recent vaccination also has been shown to be a cause. In addition there are people, very few it is true, whose sera appear to be "labile" in that they give false positive reactions without apparent reason.

In most of the above conditions the W.R. remains positive only for a comparatively short time, and tests repeated over a period of three months will usually show a weakening of the reaction or return to negativity. As a general rule, therefore, one should not diagnose syphilis on serum reactions alone unless they remain positive for at least three months. Meantime, a complete clinical examination of the patient should be carried out, including radiography of the heart and great vessels and examination of the cerebrospinal fluid.

The term "weak positive" should never be used. A "doubtful" reaction is one which is neither positive nor negative, and, except in an established case of syphilis, should merely serve as an indication for further investigation, both clinical and serological. A negative reaction does not exclude syphilis. The serum of a syphilitic patient gives a negative reaction in the first few days after the appearance of the sore, and not infrequently in the late stages of syphilis and especially in tabes dorsalis, not to say in syphilis which has been insufficiently treated.

Menopausal Obesity

Q.—*It is well known that women about the time of the menopause tend to increase in weight. One of my patients has put on several stone and is very anxious to have this reduced. I prescribed thyroid, which is fairly effective, but I am concerned as to the effect of continued doses over a long period. Is there any other line of treatment which is effective, in addition to dieting, which the patient is already carrying out?*

A.—The increase of weight at the menopause is probably associated with hypertrophy of the pituitary and adrenal glands at this time, although a great deal depends on genetic constitutional factors. There is no known "anti-adiposity" hormone yet available. Thyroid is not a specific, and even taken in doses sufficient to cause toxic symptoms it is unlikely to be fundamentally helpful. Nevertheless, it is useful in some degree, and I have not seen harmful effects from taking thyroid over a long period, providing the dose does not produce tachycardia or a pulse rate increased above normal. Dieting is helpful, but not fundamental treatment, and even on low-calorie diets the results are far from satisfactory in severe cases. Since there may be an associated element of salt-water retention, some patients respond in some degree to the additional therapeutic measure of mercurial diuretics—e.g., neptal, mersalyl, or salyrgan, 1 c.cm. intramuscularly, twice weekly, preceded the day before by 1 g. of ammonium chloride in capsule, t.d.s. Exercise and massage are also of some slight value.

Early Suckling

Q.—*I have found recently in domestic midwifery practice that many midwives and nurses are advising mothers not to put the baby to the breast until the third day, the idea apparently being to prevent too much congestion of the breasts with milk and the resultant pain and discomfort to the mother. This is contrary to my teaching, which stressed the value of colostrum, etc. Is there anything in this new idea?*

A.—There is no evidence that suckling during the first few days of the puerperium causes abnormal congestion of the mother's breasts, or that the postponement of suckling until the third day reduces any discomfort the mother may have when the milk comes into the breasts. The mother must get to know her baby and the baby must learn to suck, and what better time than the few days before the milk begins to flow? And suckling is the best stimulant to a good supply of milk. Colostrum is also said to contain protective substances and to have a beneficial laxative action. The practice of advising mothers not to put the baby to the breast until the third day should therefore be discouraged.

Hypnotics and Sulphonamides

Q.—*Which is the most suitable (a) hypnotic, (b) analgesic during treatment with the sulphonamides? (c) Is acetyl salicylic acid safe during such treatment?*

A.—The only hypnotic or analgesic drug particularly contra-indicated during sulphonamide treatment is amidopyrine. This, like the sulphonamides, occasionally causes agranulocytosis, and the combination of the two acts synergically and is very much more liable to do so. It should not be forgotten that amidopyrine (also known as pyramidon) is included in various sedative preparations known by proprietary names: these include both ethical products and patent medicines. Among the former are allonal, amidophen, cibalgin, compral, gardan, sinepan, trigemin, and veramon.

Fasciculating Muscles

Q.—*A man in the early thirties has for several years complained of involuntary contractions of muscle fibres. The contractions are identical with those fibrillating twitchings that most people have sometimes felt in the eyelids. In this case, however, they are on a larger scale and may affect any muscle in the body, but chiefly thighs, upper arms, eyelids, and quite a large number of fibres may be involved. The previous health has been good. There is no wasting but some tendency to obesity. No loss of muscular power. C.N.S. appears normal, but knee-jerks are unobtainable at times, and when present are very slight.*

A.—This is an excellent description of a rare condition in which the muscles fasciculate without any evidence of any motor neurone disease. Although it is rare the condition is well recognized by neurologists, to whom it is usually referred as amyotrophic lateral sclerosis. The prognosis is good and no disability results, although occasionally the subject will complain of a little fatigability. The only treatment required is explanation and reassurance.

Ejaculatio Praecox

Q.—*A patient aged 35 complains that for the past 2½ years he has suffered from precipitate orgasm. The orgasm itself is normal, with normal seminal emission, but always occurs as the penis is being introduced into the vagina. He is not sterile, and a child has been conceived under these circumstances. During the previous five years of married life coitus was quite normal. It is preying on his mind, the more so because his wife is showing signs of emotional stress.*

A.—It is impossible to give an adequate reply to this question without a full psychiatric history of the case. Ejaculatio praecox is a symptom of a great variety of psychoneurotic disorders from mild hysteria to latent homosexuality. Some light on the aetiology of the condition is thrown by descriptions of apparently analogous cases in married men of the same age in Dicks's *Clinical Studies in Psychopathology* (Edward Arnold, 1939). In general it has often been observed that the age between 35 and 40 is a critical one for men, characterized by disturbances of a psychosexual order. This crisis seems to be part of a process of maturing, but may bring to light previously unsuspected conflicts. Where this occurs there is only one course—namely, a consultation with a psychiatrist, preferably for both marriage partners. No rule-of-thumb remedy is at hand. In view of the previously normal function the prognosis with adequate psychotherapy is probably hopeful in this case.

Disseminated Sclerosis

Q.—*Is there any effective treatment for disseminated sclerosis? I have a patient who contracted the disease about six years ago with the usual retrobulbar neuritis. She has now much pain and weakness in the arms.*

A.—There is still no specific treatment for disseminated sclerosis. The claims which have been made for different therapeutic agents have mostly been due to the association of a spontaneous remission, which is characteristic of the disease. None of these agents, which include arsenic, serum, and vaccines, have been proved beneficial in controlled trials. There is, however, much to be done with the treatment of symptoms. This includes physiotherapy, relief of bladder symptoms, re-education in defective functions, and, most important, the maintenance of hope.

LETTERS, NOTES, ETC.

A "Gastric Board"

Capt. C. N. JEFFRIES (Army Dental Corps) writes: The object of the "gastric board," mentioned by Brigadier Edwards and Lieut.-Col. Copeman (Nov. 20, 1943, p. 640), is research into the causation of dyspeptic disorders, but to a dental surgeon the board appears to suffer from a grave omission. Consider the physiology of normal digestion: (1) a lump of food is cut off by the incisor teeth; (2) it is torn to shreds by the molar and premolar teeth, and mixed with saliva with the aid of the tongue and cheek muscles; (3) the prepared bolus is swallowed; (4) the fundus stores the bolus and surrounds it with gastric juice; (5) the pyloric antrum finishes the grinding process and mixes the food with the gastric juice. Failure of stage 2 will upset stage 5, because the food will require an excessive time to be broken down, and its surface area is too small to allow the gastric juice to act throughout the whole mass. The result is, therefore, hyperchlorhydria, not due to hypersecretion but to insufficient digestion. This process may in time lead to hyposecretion, but in any case a disturbance of function will result. Stage 2 may fail either because of the habit of not chewing each mouthful or because of loss of teeth or the wearing of inefficient dentures. Out of 217 "non-ulcer" cases no fewer than 180 were classed as "no physical disease detected (functional dyspepsia)" and only 37 had progressed to the presence of physical lesions which could be diagnosed. It would be useful to know how many of these patients had deficiencies of their masticatory apparatus. The inclusion of a dental officer in the "gastric board" may lead to some useful conclusions on the aetiology of the conditions under examination. This is a field where dentistry can be called upon to play its part, and the dental surgeon can here contribute his share to preventive medicine.

First Aid according to St. John's

Dr. BEAUMONT COMERFORD (Tolleshunt Darcy, Essex) writes: I have long wondered when a new textbook on first aid would be available. The subject-matter must often be quite unintelligible to the untutored recipients, and the lecturer himself may be a little unstable in his knowledge of these surgical catastrophes and their treatment in first aid. His cross-examining listeners may cause him to camouflage some of his answers. Dr. Truman in his letter of May 20 (p. 703) stressed that the possession of this book "is like giving a tommy-gun to a child." The first-aider may, if heroic, be a great menace to the public. When a house officer at a London hospital I saw a dead man brought in by the police: a handkerchief which had been twisted by a baton was still *in situ* on the thigh. The haemorrhage had come from a varicose ulcer of the leg. I was sorry for the constable; he was only doing what he had been told, and this tragedy had happened. At the inquest I remember stressing that the man had died of haemorrhage, but was not asked why.

Localized Postural Oedema

Dr. E. L. GAVIN (Kilbeggan) writes: I was much interested in the reply given in the "Any Questions?" column to the query on localized postural oedema (May 13, p. 697). Has any work been done on the B.S.R. in this condition? At present I have two cases very similar to those described, but in each case the B.S.R. is very high (over 40 mm. in 1 hour, micro method). In view of this, and of a previous history of vague rheumatic pains, I am treating them as cases of latent rheumatic "fever" despite any clinical evidence of this.

Two Answers

Dr. LEONARD LEY (Great Yarmouth) writes: I was interested in two of your questions in the issue of May 27. As regards coitus during pregnancy (p. 737), I am firmly convinced that this is the most frequent cause of miscarriage, and in the course of my career I have enabled numerous women to have full-term pregnancies after repeated abortion by forbidding intercourse.

The other was "a patient's dilemma" (p. 738), which amused me by the fact that it was such an excellent example of the profession's subservience to science in place of common sense. The victim suffers from nervous diarrhoea in church and is to be married, and feels sure he cannot "hold out" in church. Your correspondent deals at great length with the question but omits the obvious advice of a registry office.

Implantation Cancer

Dr. HAROLD J. SELBY (Newnham, Glos.) writes: Under the heading "Any Questions?" (May 6, p. 665) you are asked if any case has ever been recorded of cancer of the penis following intercourse with a woman suffering from carcinoma of the cervix. In view of the answer given to this question the following may be of interest. Both cases occurred in my practice. Mr. A. consulted me in March, 1933, at the age of 50, on account of a "sore" on the glans penis. He had first noticed this two weeks previously. Section proved it to be an epithelioma. He was treated with radon seeds. Two months later severe haemorrhages occurred which the patient only just survived. By October healing was almost complete and by the end of the year all was well. No recurrence has taken place and the patient is alive and well. In October, 1937, his wife consulted me at the age of 55 on account of vaginal haemorrhage. Examination revealed an extensive new growth. Section proved this to be a columnar-celled carcinoma. She was treated with radium, but only survived till September, 1938. Admittedly there is a gap of four, nearly five, years between the cure of the one and the discovery of the other; but the cervical carcinoma was extensive and inoperable when discovered, so that this gap was certainly much less than 4¾ years and possibly very much shorter.

An Old Midwives' Tale

Dr. NORMAN GOORNEY (London, S.E.10) writes: The viability of a seven-months babe and the non-viability of an eight-months have recently been discussed by your correspondents, who have whittled the origin of this down to the sixteenth century. I assure them that its origin goes back much further. It is written in the Jewish Talmud, Tractate Revomuth, page 80, and is therefore accepted as true by all learned Jews since 2,000 years and more ago when the Talmud was scribed. It is to be found for the first time in medical literature in the writings of the famous Jewish physician of the twelfth century known as Rashi.

Unusual "Deficiency" Diseases in Barbados

Dr. J. F. C. HASLAM (Director of Medical Services, Lusaka, Northern Rhodesia) writes: Dr. M. Lattey of St. George's, Bermuda, has stated in your issue of Feb. 12, p. 244, that while working in Barbados he found two conditions which he had not seen described anywhere, and he asks whether anyone else has seen those conditions. Dr. Lattey has called the first condition to which he refers "deficiency nephritis." I cannot say that I remember having seen that condition in Barbados when I lived and worked there, but I now feel fairly confident that some at least of the large amount of nephritis which occurred, and perhaps still occurs, in British Guiana may be connected with a dietetic deficiency. Many have speculated on the possible causes of the nephritis which has for long been a notable feature of the statistics of morbidity and mortality in British Guiana, and Dr. Lattey may find it worth his while to write to Dr. Wase Bailey, who is now chief medical officer of Barbados, who wrote, about 1925, a very interesting study of nephritis in British Guiana.